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EDITORIAL

I feel it a privilege to succeed Dr. Nolan D. C. Lewis as editor of The Journal of Nervous and Mental Disease. This journal was founded in 1874. It is the second oldest American journal in continuous publication in the field of neurology and psychiatry. Its long and venerable history is exemplified by the outstanding men who have guided its editorial policies and by the broad influence that the journal has exerted in its field. It has sponsored the publication of an important series of monographs, including such classics as White's *Outlines of Psychiatry*, Jung's *Psychology of Dementia Praecox*, Brill's translation of Freud's *Three Contributions to the Theory of Sex*, and *Vagotonia* by Eppinger and Hess.

From the time of its first appearance as the Chicago Journal of Nervous and Mental Disease, the editorial policy has been sensitive to the new and promising in these fields. This policy was implemented during the long editorial tenure of the late Dr. Smith Ely Jelliffe. More recently it has had the good fortune to profit from the inquisitive point of view characteristic of Dr. Lewis, from his erudition, and his incessant devotion to research and scholarship. We hope to continue in this tradition. To our new Editorial Boards we feel grateful for their willingness and enthusiasm in participating in a new and exciting venture. We hope that we will be able to sense the creative forces in our colleagues and in some measure be of help in communicating their ideas and contributions.

I consider this editorial task to be particularly challenging in these days, when new ideas and discoveries primarily in the behavioral sciences are forcing us to re-evaluate our concepts of behavior. We are compelled to take into account psychologic events that are beyond our immediate awareness and to consider social and interpersonal factors in disease. The private nature of much of the psychologic material we deal with and the special characteristics of the social phenomena involved have brought up many problems in the area of observation and data collection. In dealing with material of this kind the models of physical science do not always offer an adequate framework. The impact of the behavioral sciences has forced us into some basic consideration of epistemology. This happens, I believe, whenever new dimensions are added to our knowledge and understanding. Psychiatry above all has been the battleground where these eruptions have been most acute. One can turn away from such new directions and look back only on pain of being stultified into a pillar of salt. The struggle and the throes of change are our heritage. This is the professional tenor of our times.

The addition of new dimensions to our understanding of behavior, whether deviant or no, has of necessity brought with it the need for various approaches subsumed under the term "interdisciplinary." Productive work requires the cooperation of earnest investigators trained in different specialities, but willing to grapple with the problems of behavior. Each specialist reflects his own experience and is usually convinced of the correctness and usefulness of his own point of view in understanding the ground rules of

discovery and experiment. For productive work it is not only necessary to grope for a unifying theory, it is also necessary to tie together into a working and productive unit those who specialize in a broad array of disciplines from mathematics to philosophy —including biochemistry, psychology, sociology and anthropology.

When The Journal of Nervous and Mental Disease was founded as a quarterly by Dr. J. S. Jewell eight years after the end of the Civil War, neurology in this country was just beginning its development. Stimulated by the work of Claude Bernard and Brown-Séquard the Journal began with an interest in the sympathetic nervous system. Its early editors, especially Dr. Bernard Sachs and Dr. William G. Spiller, were instrumental in the development of American neurology and the organization of the American Neurological Association. We hope that this journal will do its share in carrying on the solid tradition of American neurology. The association between neurology and psychiatry is not an accident of history. The recent contributions to the detailed biochemistry and electrophysiology of the nervous system are pointing the way to a better understanding of behavior-brain relationships.

We have considered one of the functions of this journal to present to its readers material in the problems of neurology and psychiatry as geared to the physician working with patients. We hope that the selection of papers will be helpful to the physician. Yet it is clear that in order to integrate the clinical problems with expanding knowledge a high priority must be placed upon original research in psychiatry, neurology and in all of their ancillary disciplines. This editorial policy is reflected in the choice of our Editorial and Advisory Boards. It is our plan to publish contributions of a high order in all fields which bear upon the understanding and treatment of the patient. Our interest is in the understanding of those biologic, psychologic and social phenomena which make for illness and health.

The function of a journal should go beyond the presentation of original research contributions—granted that these have the highest priority. It should be a tool dedicated to professional education as well. From time to time our Editors will present invited papers summarizing a field, or the special interests and contribution of a given author. I should like to point to the papers of Drs. Derek Denny-Brown, Seymour Sarason and Thomas Gladwin, Heinrich Waelsch, and Hans Selye in the present issue as examples of this type of contribution. We fully anticipate that the same level of excellence will continue in the requested papers to appear in later issues. The Editors are planning a comprehensive series of current book reviews written by authorities in their respective fields. Reviews will appear in every issue, and will comprise a special section of the Journal every three months. This section will offer an extended critique of publications covering the wide range and multidisciplinary interest of the Journal. The need for transmission of important research and clinical information without delay has led the Editors to establish a section devoted to the rapid publication of brief current communications. It is proposed to publish such brief communications within one or two months after they have been sent in and accepted by the Editorial Board.

We invite our colleagues to use the journal as a live and open forum for discussion. I personally want to express my thanks to Dr. Nolan D. C. Lewis and to the staff of The Williams and Wilkins Company for their help in facilitating the editorial reorganization. I am most grateful to Drs. Grenell and Robinson for their willingness to accept a part of the responsibility for this undertaking.

JACOB E. FINESINGER

A CATARACT OF CONSEQUENCES[1]

ALAN GREGG, M. D.

The mellow assertion of Socrates that the unexamined life is not worth living provides ample basis for the broadly diastolic flavor of these opening ceremonies of the Psychiatric Institute of the University of Maryland. The diastolic period, when the chambers of the heart open to recieve the blood that is to be pumped out in the succeeding period of hard-working muscular contraction, finds its analogue in academic life in just such functions as these that Doctor Finesinger has arranged. The emphasis on such an occasion can rightly fall on receptiveness, on openess, on intake, on getting filled up—with perhaps a trivial risk of dilation—and this emphasis finds appropriate encouragement in a mood of relaxation commonly observable, if not in after-dinner speakers, then at least in their eupeptic audiences. You are, then, I trust, in a diastolic mood, ready to receive, relaxed but not empty, receptive but not surcharged, and, in some measure at least, hospitable to thoughts that you may pump later into more lively circulation.

Heartened by this gratuitous assumption, I shall venture to review some very broad circumstances in the progress of medical science, with special attention to the sequence of their development. This sequence I would call a cataract of consequences. It is but a part of the long stream of human thought, but one that much concerns us all, for we are in that stretch of the stream right now.

The cataract of consequences begins with curiosity and, in successive leaps, goes from curiosity to research, from research to knowledge, from knowledge to power, and from power to responsibility. In choosing the words "a cataract of consequences," I intentionally imply the turbulent, if not, indeed, vertiginous, character of successive changes in the current of modern Western thought and its consequences, and something, too, of its relentless and headlong force.

Why does such a review—or perhaps preview—seem appropriate? Well, for one thing, because it is often wise to look before you leap, and especially if the leap is but the first of an irreversible cataract. But more than that. The *unexamined* life of which Socrates spoke did not imply the value of examination of life after it has been lived. The word *examen* is the Latin word for the straight upright bar fixed at right angles to the horizontal yardarm of the old-fashioned scales. We read its deviations from the perpendicular not after, but during the weighing. An examination properly understood is during, not after life.

Now, the scientist as a matter of habit as well as of necessity tends to separate a problem from its surroundings and deliberately to disregard the extraneous for the sake of focusing on the relevant. One can hardly doubt that such procedure so often repeated leaves its mark on the scientist's mind. His tendency to simplify the complex has the defect of its invaluable quality, and the defect is that he tends to ignore a grave possibility, namely, that not all the actual variables get into his equation. The scientist, quite rightly, isolates his problem, only to run the risk of becoming himself isolated—isolated from some aspect of the problem that later proves inseparable from it and decisive for success or failure.

But I would suggest another reason from comprehending this cataract of conse-

[1] This paper was read by the late Dr. Alan Gregg, then Vice President of the Rockefeller Foundation, on the occasion of the Dedication Exercises marking the opening of the Psychiatric Institute, University of Maryland. Previously unpublished, this brief address clearly reflects Dr. Gregg's longstanding interest and effort in the direction of the creative in science.

quences that begins with curiosity and ends with responsibility. Essentially, the reason is that by so doing you may help to protect the interests of science and yourselves as scientists. For there is an adage which says, "The man who knows *how* will always find work: the man who knows *why* will be his employer." Obviously, the contrast is between knowing how to do something and knowing why it should be done. Scientists might well care about the question of who is to be their employer. Few can afford to be unemployed or self-employed, delightfully free as they would then be.

In the fields of science that can be cultivated only with the aid of costly instruments or complicated organization, scientists today are but rarely self-employed. Doctors are becoming less so. Rather than vainly complain that, ideally, scientists and doctors should be self-employed and thus more free, I believe it the part of wisdom for them to give more attention than they do to the task of giving the society that supports them an idea of the *why* of science. For it is those who know *why* who will support them. If you seek an example, in some ways somber and in others magnificent, an example of those who know *why* coming to be the employers of those who know *how*, regard the history of atomic energy in the last two decades. Beginning with mathematical physicists, who theorized on the how of the energy within the atom, and the experimental masters of method and technique, much of the whole enterprise is now supported and controlled by governments that believe they know *why*.

Though human curiosity, the starting point of my cataract, could alone supply enough for an hour's comment and reflection, I shall mention only two of its salient characteristics. First, curiosity is so deep and strong a characteristic of the human species that I think we shall be safe in considering curiosity as a constant in nearly all equations concerning human thought. Curiosity is not temporary or evanescent, though its object may be: curiosity itself will be continually in play, not only finding new facts but again playing on them in turn for still more enlightenment. Curiosity also possesses and confers survival value. By no mere chance did the French revolutionists close the University of Paris as an institution then dedicated to indoctrination while they left open the Collège de France, an institution devoted to inquiry and the freer ranges of human thought.

As a memory of the power of curiosity I recall a heartening episode during a visit I made once to a Mexican oil field. In the toughest part of what was euphemistically called a wide-open town, I saw, of a summer's night, a man with a large portable telescope, through which for a peso you could look at the moon. I saw that man empty a whole miserable street of gambling dens, grog shops, and the like to satisfy mere human curiosity, amid the fury and impatience of the purveyors of commoner pleasures. The scene, as I thought, called for perpetual celebration in a mural by Diego Rivera, in honor of human curiosity.

We must not forget, or even underestimate, another characteristic of curiosity; its unpredictably wide range. The varieties of human curiosity defy anticipation: one can only mark curiosity with the sign for infinity, if it is to be entered at all in the equation of human thought.

And yet, almost paradoxically, curiosity, limitless as it is, has found a strict and rigorous methodology of its own—research and its inductive reasoning. For the discovery of the research method has in effect been the discovery of the means of discovery, and research is thus both the son and the father of research. As the broad current of human curiosity is now becoming more and more channeled into research, the stream appears to quicken and gain in volume. The hundreds of millions of dollars, the hundreds of millions of hours of work now devoted to research in the Western world offer to the earlier traditional and repetitive occupations

of man a contrast—a contrast for which I can find no adequate adjective. We cannot call it stupefying, for it stimulates; nor terrifying unless we fear Truth; nor tremendous, for that is a paltry understatement. In the literary predicament of finding the right adjective, I often fall back on the advice of, I believe, Edward Everett Hale: "Adjectives are like leaves on a branch: they may make it more beautiful, but they ruin it for switching purposes." So perhaps it is best to desist from the search for an adjective and rely on the stark cogeny of saying, simply, that in comparison with known history's six thousand years of tradition, imitation, superstitious timidity, and unquestioning repetition in man's way of thinking and living, today's research actively presents a contrast. So seriously do I, for one, take the research attitude that I could regard the research activity of today as being the beginning of a new beginning in human thought, or possibly the beginning of the end.

In our cataract, the next leap, from research to knowledge, presents an almost merciful retardation. Nature answers man's presently impatient questions with a reluctance that in its slowness may be more benign than at first appears. I am not sure that it is retardation, exactly speaking. Certainly the impression is one of a reduction of momentum in the stream of human thought, but that apparently reduced momentum may well be due mostly to negative results in a large number of research efforts, no matter how briskly they started. But one can hardly say that the transition from research to knowledge is inevitably slow in every instance: it is rare, but the crucial experiment may be astonishingly quick and swiftly conclusive. And then, coming in the train of a new hypothesis or discovery, as Charles Bessey benevolently observed, there will always be those who are willing to carry back and forth "the truckloads of confirmatory observations."

The current of our cataract recaptures its speed again in the leap from knowledge to power. And lest the commonly intoxicating connotations of the word *power* egg on us eggheads into premature self-confidence, let me publicly agree with Francois Mauriac's sober remark, "A people cannot be both the most powerful and the happiest in the world." Perhaps the powerful consequences of man's recently acquired knowledge of atomic fission will again serve as an illustration—this time to etch Mauriac's lines upon our minds. "A people cannot be both the most powerful and the happiest in the world." But how does the power implicit in the control of nuclear energy compare with the power over men's minds and emotions that might be available from a comparable advance in the field of human psychology? What if psychiatrists were to learn how to split off the charged particles of emotion from the atomic units of society and so control the mass? Though I am dubious that any such fundamental advance in psychology or psychiatry is imminent, I might, by one delightful quotation, both relieve any temporary tension resulting from such a speculation and yet offer evidence that progress of a substantial sort has nonetheless really taken place, even in as difficult a field as the management of adolescent youth. The following passages, taken from the article on "Adolescence" in the Encyclopedia Brittanica, appeared in the 11th Edition, in 1910, and again in the 13th Edition, in 1927 and were only recently removed from subsequent editions. They record, one could infer, the epitome of the knowledge of adolescence available to the seekers of encyclopedic knowledge during the first third of the 20th century:

"Adolescence (Lat. *adolescentia*, from *adolescere*, to grow up, past part. *adultus*, grown up, Eng. "adult"), the term now commonly adopted for the period between childhood and maturity during which the characteristics—mental, physical, and moral—that are to make or mar the individual disclose themselves, and then mature, sometimes by

leaps and bounds, in others by more gradual evolution. The annual rate of growth in height, weight, and strength increases to a marked extent and may even be doubles. The development in the man takes place in the direction of a greater strength, in the woman towards a fitter form for maternity. The sex sense develops, the love of nature and religion and an overmastering curiosity both individual and general. This period of life, so fraught with its power for good and ill, is accordingly by far the most important and by far the most difficult for parents and educationists to deal with. The chief points for attention may be briefly indicated. Health depends mainly on two factors, heredity or the sum total of physical and mental leanings of the individual, and enviromental. In an ideal system of training, these two factors will be so fitted in and adapted to one another that what is weak or improvided for in the first will be amply compensated for in the second ...

"The residence should be on a well-drained soil, preferably near the sea in the case of a delicate child, on higher ground for those of more robust constitution. The child should be lightly clad in woolen garments all the year round, their thickness being slightly greater in winter than in summer. An abundance of simple, well-cooked food in sufficient variety, ample time at table where an atmosphere of light gaiety should be cultivated, and a period free from restraint both before and after meals. As regards the most suitable kinds of food, milk and fruit should be given in abundance, fresh meat once a day and fish or eggs once a day. Bread had better be three days old and baked in the form of small rolls to increase the ratio of crust to crumb ...

"The exercise of the body must be duly attended to. Nowadays this is provided for in the shape of games, some being optional, others prescribed. In the case of girls, let them run, leap, and climb with their brothers for the first twelve years or so of life. But as puberty approaches, with all the change, stress, and strain dependent thereon, their lives should be appropriately modified ...

"The adolescent is prone to special weaknesses and perversions. The emotions are extremely unstable and any stress put upon them may lead to undesirable results. Warm climates, tight-fitting clothes, corsets, rich food, soft mattresses, or indulgences of any kind, and also mental overstimulation, are especially to be guarded against. The day should be filled with interests of an objective —in contradistinction to subjective—kind, and the child should retire to bed at night healthily fatigued in mind and body. Let there be confidence between mother and daughter, father and son, and as the years bring the bodily changes, those in whom the children can trust can choose the fitting moments for explaining their meaning and effect, and warning against abuse of the natural functions."

Now, that is the end of the quotation, and I think it is fair to let me say so, for I am a product of that knowledge—except that my ratio of crust to crumb was probably lower, for I dearly loved beaten biscuit—and if I don't distinguish here and now between these guiding principles and myself, then, to borrow a phrase from the Encyclopedia, the stress upon my emotions "may lead to undesirable results." But is it not heartening to realize that by 1990 a digest of our present knowledge regarding child guidance or the management of adolescence may seem equally naive and descriptive—descriptive not of adolescents but of the "adults" of 1952.

I interposed this quotation deliberately to regale you in the midst of a serious effort to draw attention to how powerful research in psychology and psychiatry may become if the present rate of progress can be assumed to continue for 50 years more.

Reverting to the power that comes from knowledge, I suspect that one of the major attractions of power lies in the subtle association we assume between power and freedom. I am told that in the early history

of the Church, bishops had to take two solemn oaths at the time of their ordination. The first oath was that they would discharge the duties of that office faithfully in the sight of God and man. The second oath was called the oath of "Nolo episcopari"—"I don't want to be a bishop"—obviously a disclaimer against the charge of power-hunting, a sort of ecclesiastical equivalent of the song, "You made me love you, I didn't want to do it, I didn't want to do it." How effective a deterrent such an oath may have been remains a matter for surmise, but I take this oath as amusing and convincing but hardly necessary evidence that man's passion for power over man has been from time immemorial quite as deep as his passion for control over natural forces. Is it wise to be on guard against the power-hunters? Should the American Board of Psychiatry and Neurology, like the early Church fathers, require an oath from the aspiring diplomate that he does not *want* to practice psychiatry? I would say no, but more because I am skeptical as to the value of oaths than because I am inclined to gloss over the temptation of personal power that already confronts psychologists and psychiatrists. That temptation will increase with any increase in their skill. Lord Acton's opinion, "Power tends to corrupt; absolute power corrupts absolutely," applies starkly and gravely to psychologists and psychiatrists, whether they have chosen their profession for the sake of power or for the sake of knowledge. For knowledge moves to power. That cannot be denied or avoided, and I commend to your ruminant leisure the observation of Alfred Stern, "There is only *one* evil: to deny one's freedom in order to evade the responsibility it implies." [2]

The final stage in the cataract of consequences is the leap from power to responsibility. Responsibility to whom? Can responsibility be unilaterally defined? I think not.

To be answerable requires someone to be answerable to: to other men, to one's self, or to one's God. This triad for responsibility—to other men, to one's self, or to one's God—furnishes a swift crescendo in intangibility, unverifiability, and semantic latitude. But within this same triad of considered responsibilities have occurred some of the most agonized and noble reactings of the human spirit. Since responsibility, adequately discharged, constitutes in many ways the acme of sound social relationships of the most agonized and noble reachings of spirit, psychiatrists above all others face and must forever be preparing themselves to understand the meanings of responsibility, for so many of the disorders as well as the symptoms that the psychiatrist must handle express themselves in terms of social relationships.

Of this triad of responsibilities—responsibility to others, to one's self, and to one's God—I dare comment on but the first two.

Of the psychiatrist's answerability to man, I would reiterate that the definition of responsibility can never be unilateral. Neither intellectual snobbery nor hieratic high-handedness will escape the retaliation reserved by society for simpler types of effrontery. If a psychiatrist is answerable to other men for his decisions, he might profitably see to it, though every possible effort he can make toward their further education, that the criteria they apply to his conduct be well informed. Otherwise, as the power of psychiatry grows, dread and suspicion will await its expected tyranny. As government rests at long last on the consent of the governed, therapy depends on the implicit confidence of the patient, or better, infinitely better, on the informed conviction of the patient and of society at large. Such confidence and conviction depend upon the doctor's understanding of his full responsibility. I, for one, would not care to be answerable to ignorance, nor to be judged by the misinformed. So, if psychology and psychiatry are likely in the next decades

[2] Alfred Stern, *The Pacific Spectator*, Vol. IV, p. 392.

to pass from curiosity to research, from research to greater knowledge, from greater knowledge to greater power, and from greater power to proportionate responsibility, then the interest of psychiatry and psychiatrists calls for a substantial advance in the information needed by the laymen to whom the psychiatrist—I dare say *you* psychiatrists—will be answerable. Think of the understanding required of you in such issues as education, or leucotomy, sterilization, euthanasia, and, as anyone could see, the approaching problems of population control.

Of the doctor's answerability to himself, I can offer as a preliminary the suggestion that there might be a more widespread recognition of the difference between the Roman concept of morality and the Greek concept of virtue. The Roman was moral if his conduct was what was expected of his profession or office, his age, and his position in society. The Greek was virtuous if his conduct was consistent with his inner convictions. Quite evidently, one could in these senses of the words be moral without being virtuous, or virtuous without being moral. Nor is it all a matter of past history. If you find yourself next week on a superhighway where 65 miles an hour or upwards is expected of you by all the other drivers, you will be moral in the Roman sense if you hit that pace. But supposing you know that both your front tires are too nearly worn out to make such speed safe to yourself or others on the road. To make your conduct consistent with your convictions, you will not go over 40. You will, of course, be honked at by other drivers. But being censured by others is frequently the lot of the virtuous, as you doubtless have learned in others of lifes' pursuits.

Sometimes I wonder if the major reason for morality in the Roman sense is not quite simply this: that it provides guide lines of conduct for those who have no convictions to direct their conduct.

The whole subject of medical ethics deserves more reflection and discussion than it is getting. We are childishly naive if we think that more research will bring us more knowledge, and that's just what we want. Well, yes, but it doesn't stop there. It may go on to measures of responsibility for which we have had no preparation beyond the vague suspicion that responsibility is the heart and soul of Great Medicine.

What have I intended this evening by referring to a cataract of consequences—from curiosity that flows nowadays toward research; from research that leaps forward, at times in beautiful arcs, to knowledge; from knowledge that in a sobering parabola turns to power; and from power that cannot stay its course to eventual responsibility? Simply to suggest that men of quality, setting themselves a superior task, will reach with the inevitability of a cataract a superlatively significant destination, Responsibility.

THE NATURE OF APRAXIA[1]

D. DENNY-BROWN, M. D.

Apraxia has been defined as "inability to perform certain subjectively purposive movements or movement-complexes, with conservation of motility, of sensation, and of coordination" (57). This type of disturbance of movement has always been associated with cerebral lesions, usually of such large or diffuse nature that attempts to relate the disorder to damage to a specific area have been fruitless. The descriptive literature relating to various types of apraxia is extensive, and is characterized by a profusion of terminology that was only partly clarified by certain notable studies (27, 28, 42, 46, 53) and particularly those of Liepmann (36–38). The early history of the subject was well reviewed by Wilson in 1908 (57), and since that time by Brun in 1921–22 (10), by Woltman in 1923 (59), and by Sittig in 1931 (53). The monographs of Nielsen (44), Poetzl (48) and Critchley (11) have covered more recent aspects.

In the early literature it is apparent that the term apraxia was used by Starr, Pick (45, 46) and others in the sense that we now use the term agnosia. Liepmann's (36–38) separation of ideational from ideo-kinetic and limb-kinetic types of disorder was the first clear definition of degrees of apractic disorder of movement with and without disturbance of perception. It is not our intention to review here the many shades of opinion that have been expressed regarding Liepmann's original classification. In 1920 he himself finally elaborated a regional grouping into apractic disturbances of the limbs, usually unilateral (limb-kinetic apraxia), resulting from damage to the motor (rolandic region) of cortex, and of more complex, sometimes unilateral, sometimes bilateral, disturbances of movement without comparable disorder of perception of the nature of the movement (ideomotor apraxia) related to parietal lobe disorder. Inability to comprehend the movement, confusion of its elements, but without comparable interference with speech was ideational apraxia. This last type was always bilaterally manifested, except in specific instances of damage to the corpus callosum which result in its appearance in the non-dominant hand. The common relationship between lesions of the supramarginal gyrus on the left side with Wernicke's aphasia and ideational apraxia has been established (4, 9, 10, 28, 39, 42). In general, opinions have varied between that of Monakow who insisted that apraxia required a widespread disturbance of brain function by reason of diaschisis of interrelated function, and those of Liepmann whose more restricted scheme has not been significantly improved on. Liepmann also proposed that limb-kinetic apraxia resulted from the loss of traces of acquired motor behavior (kinesthetic memory, or engram) recording the order of components in a complex movement. Loss of a more elaborate "ideational sketch" resulted in ideational apraxia. Interruption of association between these two was responsible for ideomotor apraxia.

This paper is concerned not so much with the exact localization of apractic disorders as a new appraisal of their manifestations in clinico-physiologic terms, which we believe is a necessary first step before anatomic analysis. Our studies of parietal lobe disorder have convinced us that disturbance of awareness of events in one side of the body

[1] From the Neurological Unit, Boston City Hospital and the Department of Neurology, Harvard Medical School. A short version of this paper was presented to the Ninth Annual Meeting of the American Academy of Neurology, Boston, 25th April, 1957. The photographic expenses of this investigation have been defrayed by the Harrington Fund.

and of space may be present in varying degrees, associated with release of involuntary motor reactions that interfere with the use of the limbs. From this point of view many of the original observations of Liepmann (36–38) and others are open to different interpretation. It becomes apparent that in situations where defect in perception was previously assumed to be absent, or not significant, we would now recognize such a defect. Conversely, disorders such as the grasp reflex, considered by Liepmann and by Wilson and Walshe (58) to result from perseveration of ideation, became later identified as a motor reflex. Our own studies indicate that this conception must now in turn be revised. It is also apparent that earlier views regarding the function of the corpus callosum are untenable.

The following account attempts to relate the various manifestations of apraxia to a physiologic disorder of movement as a whole. In order to keep within physiologic terms it is necessary to maintain an approach as objective as possible, avoiding postulates such as "kinesthetic images" that cannot be verified by observation.

IDEATIONAL APRAXIA

When apraxia results from some type of diffuse lesion, for example a degenerative brain disease such as presenile dementia, the defect is first seen in relation to complex movements performed by request of the examiner. It is then obvious that the more hypothetic the nature of the request, the more imaginary the circumstances, and the more the requested movement is a mimesis of the real thing, the more vulnerable it is to such disease. The patient is unable to *pretend* to drink from a glass of water, or even to pretend to drink from an empty glass (Fig. 1, A), yet can perform without difficulty when presented with a glass with water in it (Fig. 1, B). This is the distinction between the vulnerability of propositional performance versus spontaneous or emotional performance that Jackson (27) pointed out in relation to disturbances of speech, Pick (45, 46) and Liepmann (36) in relation to apraxia, and applied directly to abstract and concrete behavior by Goldstein (22). The same differential loss is seen when the patient cannot name a pencil or suggest a use for it when presented with it, yet a moment later picks it up from among many other objects and uses it when given paper and asked to write something down (Fig. 1, D). The first involves a conceptual sequence involving the symbolism of names and visual imagery, the second is a sequence of behavior that involves automatic motor activity in relation to usual environmental stimuli. The distinction is between the object and related performance as a mental (psychologic) exercise, and the object as part of a physiologic performance.

In such a defect the loss is clearly the conceptual process, and this is what we understand by ideational apraxia.

In the presence of less diffuse, more localized lesions ideational apraxia may be restricted to one or other general category of the perceptual defects peculiar to agnosia. It is then even more clearly related to the perceptual defect. For example in the "constructional apraxia" of Kleist (28) and Mayer-Gross (40, 41) there is defective copying of models, whether of Koh's blocks or of drawing. The patient appears to have difficulty in carrying the pattern in his mind when looking from the model to his own construction, keeps trying to bring his construction nearer to the pattern, yet still cannot copy the details, as in Dr. Alexandra Adler's (1) patient studied in our clinic and shown in Figure 1, E, F. This defect is also related to a highly propositional percept, and involves other aspects of spatial perception. It illustrates in addition the difficulty in defining recall memory and "engrams" in relation to any motor performance. Whereas the usually accepted definition of engram implies a cortical trace established by ha-

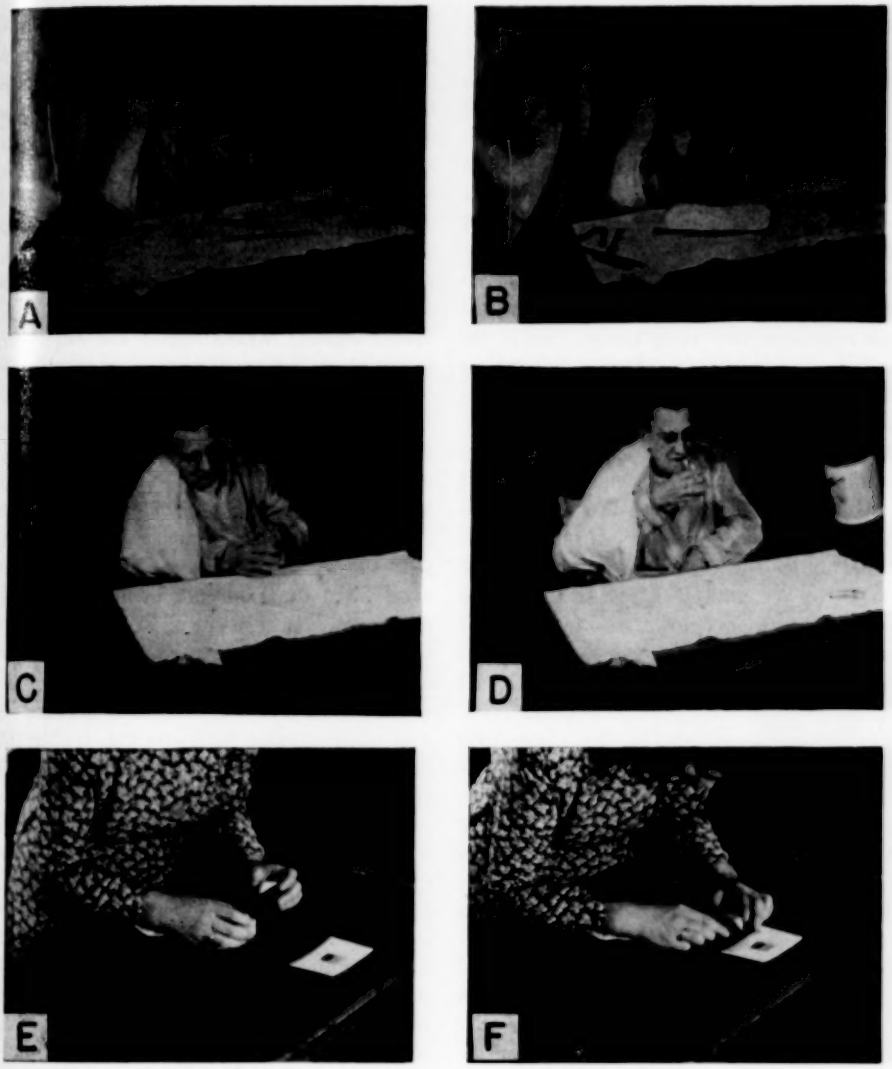

FIG. 1. Ideational apraxia. A patient suffering from hypertensive cerebrovascular disease in A is unable to identify any of the objects on the table including a pencil, for which she cannot suggest a use, yet in B, when given some paper and asked to write, picked up the pencil and used it naturally. In C she cannot name or suggest a function for a glass, yet in D spontaneously drank from it when it was filled with water. In E another patient (reported by Adler) shows constructional apraxia with Koh's blocks which she ineffectively in F moves closer to the requested pattern.

bituation, defective *formation* of the pattern would appear to be inherent in the perceptual difficulty in constructional apraxia.

More laterally placed lesions result in finger agnosia, and in more severe form, difficulty in identification of nose, tongue and face in the patient or on another person. This also is associated with behavioral defect in

the nature of inability to exhibit facial expression to order, with less restriction of spontaneous facial expression. Inability to gesture with the limbs has been commented upon by Wilson and others in association with loss of facial expression. Again it is as if the patient was unable to formulate a mental pattern of himself at the moment of testing.

Following Schilder (50), it is customary to ascribe such apraxia to loss of a psychologic reference, the "body image," for if the patient appears to have difficulty in forming any conception of his own anatomy he also cannot perceive what is requested of him. The "body image" is an extension of the postulate of the "postural scheme" proposed by Head in explanation of loss of position sense. An extension of both postulates is included under the term loss of "kinesthetic image" to account for apraxia. As will be discussed further below, loss of sense of position is not necessarily associated with apraxia, and may be severe in degree when identification of body parts is not disturbed. The agnosia responsible for face-limb apraxia appears to involve a complete conceptual inability, as if bodily configuration could not exist as a proposition. Similarly in constructional apraxia the geometric pattern cannot be formed as a propositional percept. Lhermitte (32–35) sees a disorder of "la pensée spatiale" as the basic fault, with subdivisions of disorder of body image and representation of extrapersonal space. Such terms describe the two chief areas of disturbance, but we find no evidence to identify them with postulated functions. We fail to find evidence that disorder or loss of body image results in limb apraxia, or that loss of a postulated "spatial sense" results in constructional or right-left apraxia. From the physiologic point of view we prefer the alternative hypothesis that usually *both* a specific conceptual function *and* its related motor performance are disturbed together in relation to spatial configuration either of external objects or of persons.

In some patients the disturbance of function may be greater in degree in the conceptual aspect, in others in the related performance. For example, the concept may be denied yet the performance unimpaired. Such difference in accentuation is related to other extraneous, interfering factors that will be discussed separately. Aphasia, in the sense of use of names of objects in speech, reflects the same type of conceptually dependent motor difficulty but with minimal visual aspects, which confer the chief differentiation between apractic speech disorder and other types of apraxia. As Head (23, 24) has noted, aphasic difficulty can be assisted by mimicry (facilitation by visual means) whereas agnostic apraxia is not aided in this way. The patient with apraxia may be incapable of touching the right ear with the left hand when asked to do so, and even when the examiner demonstrates the movement, yet as in one of Brun's (10) cases will immediately raise the hand to the ear in annoyance if the examiner pinches it. In our experience he will complain that his ear was pinched, and therefore can under this circumstance identify the part. Alternatively, if his understanding of the spoken request is facilitated by example and it is more clearly only the spoken words "ear" or "finger" that have no meaning we then call the disturbance sensory (amnestic) aphasia. We therefore find such disorder more comprehensible in terms of specific organization of receptive patterns of incoming stimuli from the environment than in relation to loss of a postulated standard such as "the body image."

Although the complex movements of lips, tongue and larynx show disturbances that are apractic in nature, these occur in isolation from other types of body apraxia, as if the praxis of speech had developed independently in the dominant insula and parietal operculum, more removed from the parieto-occipital region concerned in other types of ideational apraxia. Thus apraxia of the tongue is a special and particular variant

of motor apraxia, associated most commonly with executive aphasia, and usually dissociated from apraxia of facial expression.

ADEXTROUS APRAXIA

Occasionally, the propositional, purely ideational objective of a motor performance is completely dissociated from the physiologic by reason of management ("dominance") by different hemispheres. The symbolism involved in writing is often dissociated from that of reading, but the symbolism of words can sometimes be separated from the motor performance. For example, we have reported elsewhere with Chambers (15) a left-handed patient who had learned to write with his right hand and who developed a right parieto-occipital lesion. He then could go through all the movements of writing with the right hand, yet was unable to form an intelligible word. The associated denial of disability led him to believe he was writing intelligibly. He had retained a right-handed skill for which the symbolic ideation was lost. The strongly right-handed man with a left hemisphere lesion causing alexia cannot make even a pretense of writing because he has lost both the skill and the ideation. In the same way a right parietal lesion in a right-handed patient commonly leads to inability to complete dressing himself on the left side. A left parietal lesion of similar kind destroys the whole skill of putting on a garment, so that the one-sided failure is then seldom seen, though with Banker (14) we have recorded one such case.

The type of dominance that can account for failure of the mechanism for psychologic usage of symbols such as words, names, numerals and spatial patterns as a result of one-sided lesion is therefore not necessarily related to the same hemisphere that dominates the initiation of learned skills that require a leading hand. Thus inability to play a musical instrument need not be associated with difficulty in reading music, to write with reading defect, to tie a knot with loss of visuo-spatial sensation, if motor and symbolic dominance are on different sides. Such motor loss is a cortical motor defect that can be compensated only by relearning the special skill with the other hand. A subcortical lesion is followed by immediate ability to use residual motor function, clumsily at first but with the general pattern of movement intact. With a cortical lesion the whole skill is lost, so that the patient cannot perform even a rudimentary pattern of the movement complex.

We would call this element of apraxia "adextrous" apraxia in order to distinguish it from other types we shall discuss below. It will be noted that lack of dexterity should be apparent in movements such as cutting with a knife, buttoning, even striking a match, as well as more specialized performance such as writing. Difficulty in writing without comparable loss of ability to read from left hemisphere lesion in a right-handed person would appear to be adextrous apraxia and it is then often assumed that a postulated "reading center" has been present in the right hemisphere is such a case. Since the ideational component of such apraxia is not readily apparent, such disturbances have in the past been attributed to loss of association pathways, notably the corpus callosum. Akelaitis has shown that apraxia does not follow division of the corpus callosum. The outstanding lesson from the observation of cases of hemispherectomy is that the hemisphere responsible for "dexterity" can be removed with ultimate restoration of dextrous performance on the other side as in the patient reported by Bell and Karnosh (6). Whatever the corpus callosum may contribute, it is not in the coordinated action of both limbs in the synthesis of a motor act, but in some relation to priority of sensorimotor initiation of behavior by one or other hemisphere. It is notable, for example, that the studies of strychninization by Bailey and his associates (5) showed callosal connections to be predominantly related to areas 6, 7, and 18, two of which when stimu-

lated inhibit automatisms in the lightly anesthetized animal, as if a cortical sensorimotor activity on one side might suppress any comparable reaction on the other. It therefore appears that investigation of cases where mirror behavior of the two opposite limbs could not be suppressed, the "bimanual synergia" of Fragstein (20) and Drinkwater (17), would be of interest in relation to agenesis or lesion of corpus callosum. Thus neurophysiology would seek an explanation for bilateral reactions in terms of adequate stimulus rather than in terms of bilateral organization of motor "centers." Each cerebral hemisphere, independently of the other, appears to be able to initiate bilateral behavior without direct participation of the other, though the detailed performance of the ipsilateral limb requires the integrity of its corresponding hemisphere.

The most difficult problem in the group of adextrous apraxia is that where writing is lost or impaired in a right-handed person as a result of right hemisphere lesion, without loss of reading ability. Leonhard (31) has recently returned to this problem pointing out the defects of a postulated right hemisphere "writing center" in a right-handed person, and returning to Liepmann's concept of interruption of a callosal association pathway. Since this interpretation is denied by the work of Akelaitis (2), the only other present alternative is that of Herrman and Poetzl (26) that dexterity is dependent upon constructional apraxia (a bilateral defect from unilateral lesion). Constructional apraxia is a destruction of the ability to reproduce written patterns as well as designs of blocks or matchsticks, and has been found as often from right hemisphere lesions as from left (11) yet need not interfere with reading. In this case the defect would be ideational, but it still implies that bilateral or ipsilateral movement initiated by a dominant perceptive mechanism operates at subthalamic level. It is also clear that more studies of the relationship between constructional apraxia and dexterity are needed before we can be certain as to what determines motor dexterity and its loss in particular circumstanes.

APRACTIC PERSEVERATION OF IDENTIFICATION

Perseveration is prominent in relation to most cortical disturbances of speech and motor behavior. Its prominence in apraxia has been emphasized since the term was introduced by Neisser (43). Pick (45, 46) pointed to the repeated elements of some previous motor performance that lead to apraxia when a new act is requested, and maintained that such apraxia was not necessarily associated with loss of sensory images or memories. Such perseveration as occurs when for example a patient attempts to comb his hair with a cigar when previously he had been shown a comb, clearly indicates an abnormal persistence of ideation. It is commonly stated that such perseveration occurs in the absence of any disorder of perception, and is therefore ideokinetic apraxia. We would maintain that there is always some defect in perception in such circumstances, and that perseveration is a release phenomenon in relation to partial damage to cortical perceptive mechanisms. Thus in the above instance there should have been difficulty in obtaining the recognition of a comb in the first place, following which comb and comb-function perseverated either consciously or unconsciously. In our experiments with monkey cortex reported elsewhere with Chambers (15) we have related how animals with opercular parietal lesion react violently to pin prick long after the stimulus, and again in subsequent minutes without further stimulus. After upper parastriate lesion the animal will look down towards a moving object in the lower visual field, continue to do so long after the stimulus has gone, and again repeatedly in the next five minutes without further stimulus. The converse is demonstrable in lower parastriate lesions, the animal then being found to look up frequently without fresh stimulus. After post-central gyrus lesions body con-

tact righting responses result in unusual postures that persist long after the stimulus is removed. In all these instances partial ablation of receptive cortex results in raised threshold for the appropriate type of stimulus, with a prolonged "after-discharge" of the residual cortical sensory mechanism.

In this sense perseveration is a sign of perceptive disorder, and its associated behavioral (motor) response comes into the general category of ideational apraxia. Whereas the types of ideational apraxia that depend on specific agnosias relate to loss of recognition of specific symbolic categories, perseveration in any of these categories indicates partial disorder. Since partial disorders are more common than complete discrete agnosias, perseveration of motor acts is the most common type of ideational apraxia.

The types of apraxia we have described as adextrous apraxia and perseveration of identification are usually classed as ideokinetic apraxia. We doubt the absence of disturbance of ideation in such types and would advocate dropping the confusing word "ideokinetic."

UNILATERAL APRAXIA

If the true agnosias are dependent upon a "dominant" function in relation to the symbolic currency of mental imagery and speech they should relate to behavior in any part of space or person. Apraxia that is secondary to agnosia should therefore occur in both the right and left hand. We made a sharp distinction between true agnosia and the perceptual disorder resulting from right parietal lesion in a right-handed patient, which is usually a defect in recognition of a lower physiologic level than agnosia. In 1952 we suggested the word "amorphosynthesis" for this unilateral type of failure of recognition, and have pointed to its relative character, resulting from perceptual rivalry. It is manifest chiefly in terms of behavior such as that described by Brain (8) and Hécaen (25) and others as the "apraxia of dressing" that we regard as the result of shrinkage of the biologic significance of stimuli arising in the related half-field. The symbolic aspects of perception are not disturbed. The patient may fail to use or even to recognize his left arm, but he is well aware of its parts, can identify them correctly when requested, or identify corresponding parts in a model. In spite of loss of position sense the essential elements of a complex movement can be performed to order, though another type of defect usually called ataxia may interefere with performance. By Foerster's (19) definition apraxia is not then present because the prime mover is correctly used. If a certain performance such as dressing one limb, brushing one side of the hair, shaving one side of the face fails to appear, it is not on request for that particular performance but only as part of more general behavior whether spontaneous or commanded. The difficulty here points to a one-sided defect in the biologic necessity for movement and not to inability to formulate a behavioral proposition. This type of behavior is associated with perceptive defect such as estimation of two-point sensation, or tactile localization, and in the corresponding visual sphere defective estimation of the mid-point in a line or figure. We prefer to call the associated behaviorial defect neglect of one side of person and of space rather than apraxia, for truly purposive movement may be undisturbed.

In addition to the behavioral defect in relation to unilateral parietal lesion there is frequently an abnormal, involuntary attitude or movement that appears to be an unconscious low-level release phenomenon. The patient with failure to dress one side and amorphosynthesis presents an abnormal reaction to contact with the hand that we have called "the avoiding response" (Fig. 2), and sometimes catatonic persistence of attitude in the limbs of the affected side. This type of disorder of movement is localized to one limb, or one side, and we shall discuss

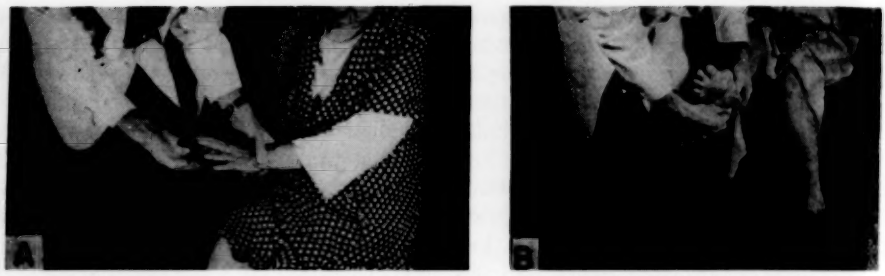

FIG. 2. A. Extension and spreading of the fingers, with extension of the wrist ("instinctive avoiding response") elicited by light stroking of the palm of the hand from palm to fingers. B. The avoiding response of the foot.

it further as kinetic apraxia. Although symbolic imagery and dextrous function may also be disturbed when the lesion is in the dominant hemisphere the essential elements of neglect and avoiding are independent of these.

KINETIC APRAXIA

Liepmann (36–38) and Wilson (57) were well aware of the condition that was later called forced grasping by Schuster (51), or by us instinctive grasp reaction resulting from frontal lobe lesion, and classed this as a type of limb-kinetic apraxia. The contrary condition resulting from parietal lobe lesion we have termed "the avoiding reaction" or instinctive avoiding reaction is a similar type of response. Grasping and avoiding are incompatible opposites, for the stimulus that leads to closing of the hand in the one causes it to extend in the other. The response, however, is not limited to the hand. In association with forced grasping the sucking and pursuit movement of the head when the lips are touched (Fig. 3, A and B) is paralleled by retraction of the lips and adversive movement of the head with avoiding (Fig. 3, C). Forced grasping is only one aspect of a total change in behavior, towards a compulsive exploration of the environment. Yet in terms of each part of the compulsion that is examined, it is a reaction to stimulus, tactile or visual, that leads to exploration and prehension, whether by hand or lips, and in less degree by the foot. If the attention of a normal person is directed elsewhere, contact with the hand elicits either no response, or a slight flexion movement of the terminal phalanges if the stimulus falls in a limited area on the palmar surface of the first phalanges. If the stimulus falls on the dorsal surface of the last two phalanges, a slight extension of the fingers may result. When instinctive grasping is present, the area eliciting flexion

FIG. 3. A and B illustrate the approach and pursuit movement of head and lips associated with instinctive sucking, in a patient with strongly developed instinctive grasping in the right hand. C illustrates the turning away of head and spreading of the lips in association with avoiding responses in the limbs.

is greatly extended to include the whole palm as shown by Seyffarth and Denny-Brown (52). Touch on the dorsum now elicits an orienting movement to bring the stimulus into the palm. Conversely the parietal lobe disorder shows a retraction from all forms of environmental stimuli. The normal reflexogenous area for extension of the hand now extends onto the palmar surface of the hand leading to withdrawal from a stimulus that normally causes slight flexion, and only deep pressure over the palmar surface of the first phalanx can initiate grasping. Similarly the normal reflexogenous areas of the face and foot shrink or extend in relation to these two types of lesion.

Let us now examine ability for movement in the presence of these two states. In the patient with *frontal lobe lesion* the prominence and persistence of instinctive grasping constitute a major disturbance of motor coordination. The patient may not be able to relax his grasp on the bedclothes or other object. The grasp continues because the overactive effect of the stimulus to the palm persists. The hand actively pursues the stimulus (Fig. 4, A, B). If the patient can escape from the stimulus he can relax the hand. Yet, if we examine the use of the hand carefully, we can observe that its use is very awkward and clumsy even when there is nothing in contact with the palm. The patient does not open the hand wide enough when he takes hold of an object; he tends to advance it too late, and then too far. He cannot open and close it quickly, even when the hand is empty, and the defect here is slowness in the opening phase. His performance in any rapid movement is limited by this slowness and incompleteness of extension. Likewise

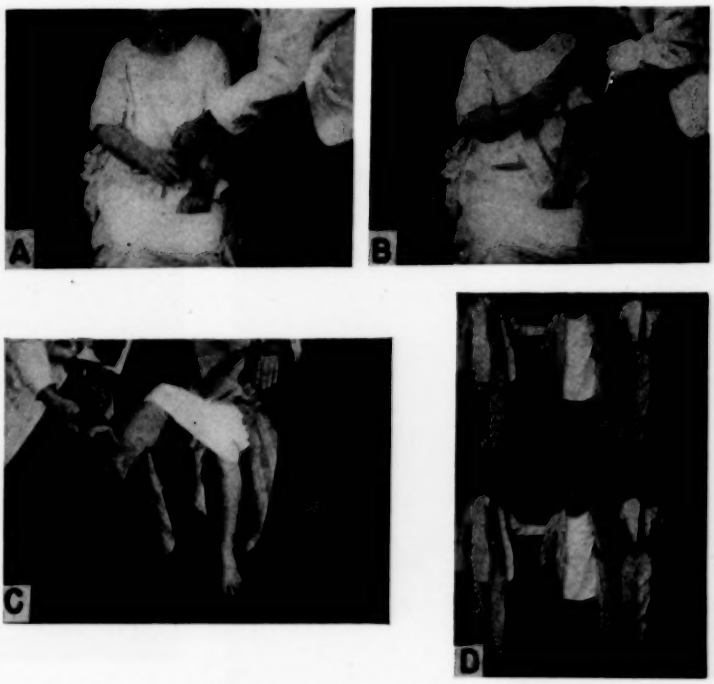

FIG. 4. A and B show the instinctive grasp reaction and pursuit of the retreating stimulus in the right hand in a patient with left frontal tumor. C shows the toe grasp in the foot. D shows one small shuffle of the left foot as a patient with carotid syndrome with grasping in the left limbs, without hemiplegia or spasticity, attempts to walk.

the movement of his lips is slow and incomplete. When the hand makes contact with any object, even if not yet grasping, the whole limb tends to stiffen. Even a passive contact with the limb has this effect, so that on manipulating the arm of the patient when holding the hand, the examiner feels a stiffening of the limb on every applied movement, the "gegenhalten" or "negativism" of frontal lobe lesions. This stiffness is less or absent if the examiner holds the limb by the wrist. When the patient attempts to write, the hand stiffens and appears to stick to the paper. When severe this is the apraxia of writing of frontal lobe lesions. A foot grasp may be present (Fig. 4, C). When the patient then puts the affected foot to the floor, the lower limb stiffens, becomes glued to the floor so that steps are made only with great difficulty, and when made, are each a brief shuffle (Fig. 4, D). Only when the heel is at last raised from the floor can the foot be lifted and a complete step made. Even then the toes fail to extend completely as the foot is advanced. Yet the lower limb can be moved freely in space. With bilateral frontal defect in motor function this difficulty in making the first step becomes very obvious (Fig. 5), a rapid shuffling of feet before a step can be made (we call it the "slipping clutch" syndrome). The patient leans forward and flexes the elbows. Yet the same patient can make the movement without difficulty when the limbs are free in air. This can occur in the complete absence of spasticity, and with flexor plantar responses. Foot grasping is present, and in walking the toes are seen to flex strongly.

We therefore see that as a result of mesially placed lesions of the frontal lobe, all types of movement are interfered with by a mechanism that in all its essentials is exemplified by instinctive grasp—namely, an abnormal persistence of posture, including grasping in the hand, compression in the lips, and standing in the foot, in response to contact, with overemphasis of flexion of the fingers and toes, tensing of the lips, throughout all types of movement. The disorder also exhibits perseveration, for the defect is increased by any contact reaction and persists thereafter for a time in movements made

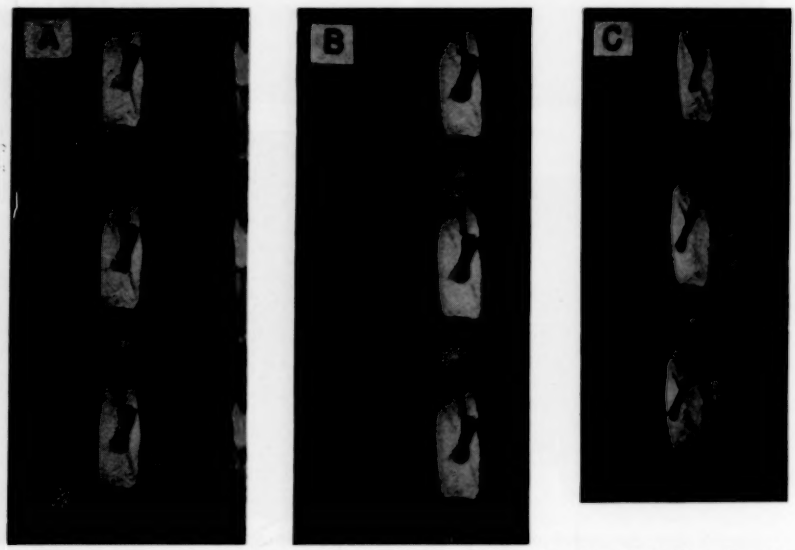

FIG. 5. Three moving picture sequences showing in A and B a series of small shuffles of one foot, then the other, as a patient with bilateral frontal lobe degenerative disease attempts to walk. Eventually, in C, he gets one heel, then the other off the ground and can make natural steps.

freely in space. This type of disorder may be manifest in various degrees, from slight reactions to moving contact with the lips or palm of the hand that can only be distinguished from normality by comparison with the unaffected side, to such intensity of bilateral reaction that the patient cannot walk or reach out, yet the plantar responses remain flexor in type. When the condition is severe the toes tend to plantar flexion as a result of all stimulation except pain.

In contrast let us look more closely at the movements of a patient with *parietal lesion*. The wrist and fingers tend to extend in response to any form of tactile stimulation, particularly to a distally moving tactile stimulus to the palm of the hand (Fig. 2). Rarely the phenomenon is so remarkable that the fingers and wrist (Figs. 6, 7) and even the whole arm, may extend as the examiner approaches ("levitation"). If the patient can use the hand to grasp an object, it is seen that the fingers and wrist extend to an abnormal degree as the object is approached (Figs. 6 and 7), giving an impression of exaggerated and unnecessary movement. Even in finger-nose-finger test, the patient's hand hesitates before touching the examiner's finger, but not his nose. The closure of the hand on grasping is too light, and is relaxed to easily so that the object often drops. The overextension of fingers, and consequent difficulty in fitting them to a pair of scissors, for example, is particularly instructive (Fig. 8, C). Yet the patient can use the scissors correctly as in the illustration in Figures 8, D. It is dif-

FIG. 6. A shows the spontaneous (perseverating) avoiding posture of the left hand when the patient with right parietal lesion (16) is directed to hold the hands outstretched. In B the overextension of the same hand during movement is seen as the patient grasps the examiner's hand. In C another patient with similar right parietal vascular lesion shows the same perseveration of avoiding. In D when asked to walk she rose with difficulty and advanced the left foot forward with overflexed knees and extended toes, tending to fall backward.

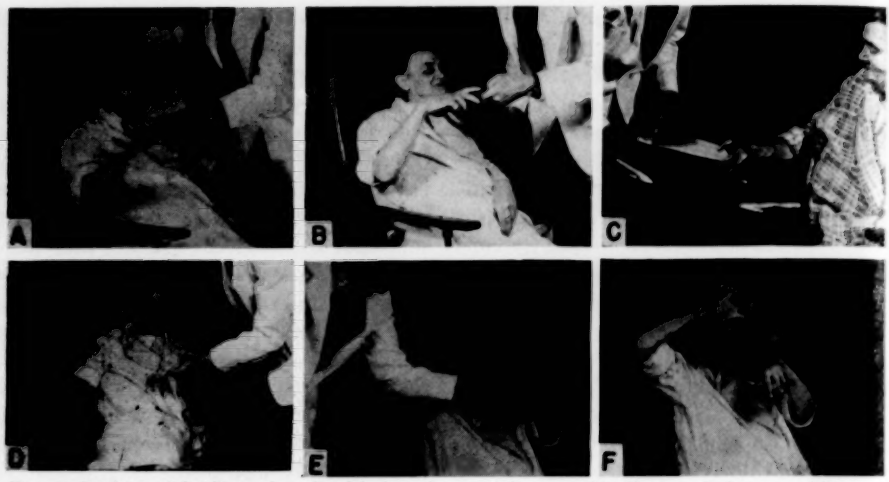

FIG. 7. Patient with bilateral parietal lesions from cerebrovascular disease. A shows avoiding reaction in right hand, levitation in left. B shows overextension when told to grasp examiner's hand, and C when picking up object. D shows levitation in the right hand when the left was manipulated. E shows attitudes imposed by handling the limbs, and F catatonic persistence of posture approximately two minutes later.

ficult for the patient to initiate the use of the hand, and he will often reach over and use the other hand at the last moment. Such a patient putting on spectacles will use the normal hand to put them on, the affected hand going through partial movements at a distance from the spectacles on its own side, but not making contact with them (Fig. 8, A). There is not only a tendency to withdraw from any contact but a difficulty in making contact. The limb is too withdrawn, the fingers and toes too extended in all phases of movement. When the condition is severe, the patient cannot move one finger of the affected hand without overextending the others even with the eyes open (Fig. 7). When the patient attempts to write with such a hand, contact is made with the paper only for brief intervals of time, and the hand intermittently withdraws from the paper (Fig. 8). Touching the lips leads to opening the mouth and turning away of the head (Fig. 3). In walking the foot showing avoiding responses is lifted too high, and contact with the ground brief (Fig. 6, D) with overextended toes. The toes are often held extended for long periods (a "permanent Babinski sign") and stimulation of the sole of the foot increases this.

Also in direct contrast with the effects of frontal lobe lesions, the limb is found to be loose, hypotonic, when manipulated by the examiner, yet when he releases it in any posture it tends to remain in catatonic persistence of that attitude. The abnormal everted and overextended posture of the affected hand when outstretched (Fig. 6) that we have used as a useful clinical sign of parietal lobe damage, appears to be a perseveration of an avoiding reaction induced by some previous contact. Catatonic persistence of imposed attitudes is prominent when there is bilateral parietal lesion. The gait is then reduced to shuffling advancement of the foot in front of the patient, who tends to fall backward with toes overextended. He makes inadequate attempt to correct displacement of center of gravity. Yet he can make all the requisite movements if the limbs are freely suspended in space. The patient appears to have difficulty in approaching a chair, turning and sitting in

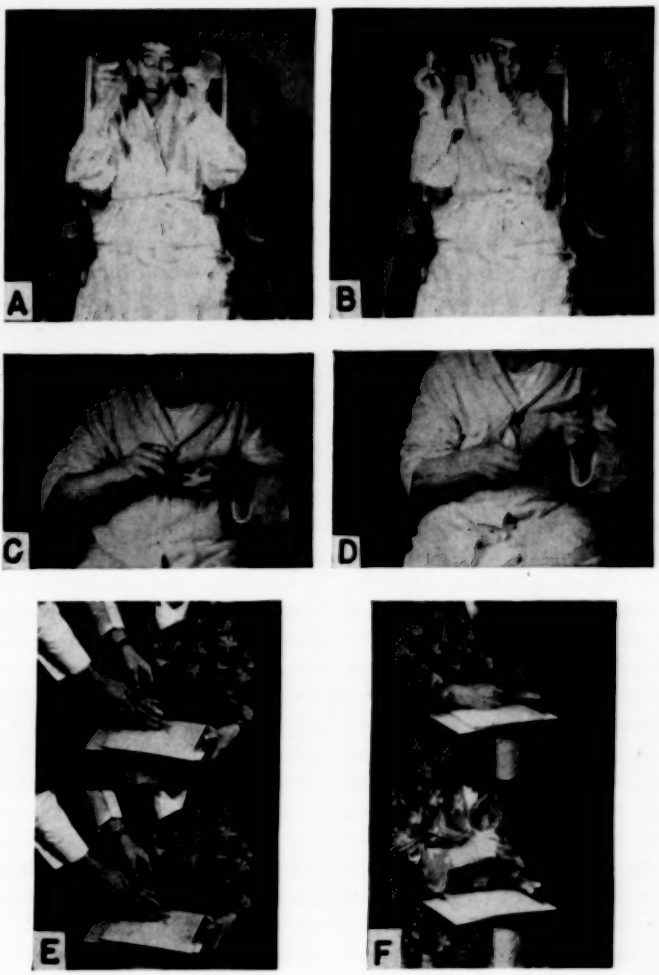

FIG. 8. A and B show patient with left parietal lesion putting on spectacles. The right hand, which gave strong avoiding reactions, performs associated movements without touching the spectacles. C patient with left parietal lesion unable to fit the fingers of the right hand to a pair of scissors, yet in D can use the scissors well when they have been fitted to the fingers. In E a patient with similar lesion and strong avoiding has difficulty grasping a pencil. When it is successfully grasped, the patient can begin to write (F, upper) but writing is intermittently interrupted by a sudden flexion of elbow and extension of fingers (F, lower, consecutive movie frames at 16 per second).

it. He may spend long periods making tentative and useless approaches to it [the "trunk apraxia" of Sittig (53)]. When lying he may have great difficulty sitting up. The corresponding perseveration of adversive movement of the mouth and head is more seldom released. It may possibly require a more extensive lesion, for it is seen in post-encephalitic states.

As with abnormal grasping responses, the avoiding reactions may be present in various degree. Slight brief upward movement of the fingers in response to a distally moving light contact with the palm of the hand, or

of the toes to similar stimulation of the foot, is not uncommon in normal people. An early pathologic response can only be estimated by its greater intensity in comparison with the normal side, and by its regularity of exhibition. A heavy moving contact in the palm of the hand may then still elicit a grasp response, the "coarse" grasp reflex of Seyffarth and Denny-Brown (52). Similarly in the foot, when moderately facile avoiding can be elicited by light stroking, a heavy distally moving contact of the type used to test the plantar response will still evoke a normal downward movement of the great toe. When prolonged dorsiflexion can be regularly obtained by light moving contact, the associated apractic disorders of movement may be expected.

The grasping response associated with frontal lobe lesion is frequently clonic in nature, and this tremor appears in all movement in the same limb. In the outstretched hand, a brief moving contact can usually initiate a brief flexion, followed by several rapid alternating flexions and extensions. With parietal lesions tremor is less frequent, but when present is a rapid alternating tremor of wrist and fingers at rest, increased and spreading to proximal joints during movement. This tremor can also be initiated by eliciting an avoiding response, which then forms the first beat of an alternation for several beats. The initiation of tremor in this fashion, particularly when it has not been evident in other movements, points to a critical degree of instability in the opposing grasping and avoiding factors in the regulation of movement.

We therefore distinguish two contrasting types of kinetic apraxia, the one traceable to perseveration of all contactual reactions, the other to perseveration of all withdrawal responses. Since these disorders are much more comprehensive than their prototype reactions, instinctive grasping and instinctive avoiding, we term them frontal or *magnetic* apraxia and parietal or *repellent* apraxia respectively. Together they represent defective kinetic projection of behavior. They are common disorders and even in moderate degree they interfere seriously with motor performance. In a recent paper with Chambers (15) we have discussed the experimental evidence to show that the magnetic, exploratory aspect of behavior in relation to the environment is managed by the cortex of the parietal lobe, and released by frontal and temporal lesions. Conversely, the repellent, negative bias to behavior is determined by a strip of cerebral cortex in the premotor, cingulate and hippocampal region and released by parietal lesions. We believe that these two types of response represent two areas of normal organization, positive and negative, of the tropisms to the environment managed by the cerebral cortex. Damage to the mechanism of either one releases abnormal activity of the other (13).

Magnetic and repellent types of apraxia are most commonly unilateral, resulting from lesions of one hemisphere. As a result of degenerative lesions, particularly those with chiefly frontal or chiefly parietal incidence, kinetic apraxia may be the most prominent clinical feature. When these disorders are bilateral, the bilaterally coordinated movements such as those of walking, rising, sitting are the first and most severely affected. Complete immobility except for sucking and biting movements may be the end result, yet still without the bilateral spastic element that defines pseudobulbar paralysis. Spastic paralysis is customarily defined separately from all such apraxias, though various mixtures of spastic and kinetic apractic disorder commonly occur.

The disturbances of movement we have grouped together under kinetic apraxia appear to us to include all the types of "limb-kinetic" apraxia described by Liepmann (36–38), some types of ideomotor apraxia of Liepmann, the unilateral motor and sensory and some bilateral motor forms of Brun (10), and the trunk apraxias of Sittig (53). The avoiding reactions that determine repellent apraxia include the type of difficulty in

handling objects described by Poetzl (47). All types of kinetic apraxia are to be sharply distinguished from the motor disorders traceable to absence or perseveration of the propositional aspects of perception.

CEREBRAL PARALYSIS IN CHILDREN

The development of integration of movement in the young infant exemplifies the successive acquisition of withdrawal reflex, reflex grasping, instinctive avoiding and instinctive grasping, leading to the delicate balance of reactions that constitutes motor normality. Cerebral lesions in young children interrupt the natural sequence of development, leaving a state in which motor behavior is dominated by one or other of these modes of reaction. Instinctive avoiding is the most prominent of these arrested stages, commonly in unstable equilibrium with instinctive grasping, the result being the fluctuating state that is called athetosis. In the early stages after a cerebral illness in infancy, particularly when bilateral hemisphere damage has occurred from encephalitis or lead encephalopathy, the development of either repellent or magnetic syndrome may be very striking. In repellent apraxia the limbs may withdraw consistently from every contact, and collapse every time they make contact with the ground, yet be stiffly extended when free in air (Fig. 9, A and B). In these circumstances the labyrinthine righting reflexes are very brisk, and appear to be responsible for the stiff attitudes when suspended in air. The child shown in Figure 9 was unable to walk or stand because of these reactions following encephalitis. Avoiding of remarkable degree followed any contact with hands, feet or face (Fig. 9, C). Magnetic apraxia may be equally prominent in the young child with cerebral paralysis from birth injury, where stiff extension of the limb on contact with any surface may make the otherwise limp child unable to walk. In such cases magnetic and grasp reactions may be very intense (Fig. 9, D, E). In the child recovering from hemiplegia or transverse spinal lesion fragmentary behavior of the same type is often found to complicate spastic paralysis, but we wish here to emphasize that the pure apractic syndromes may acount for inability to use the limbs, in the absence of spastic paralysis.

SUBCORTICAL APRAXIA

It is characteristic of the effects of cortical lesions that the released, overactive cortical reaction should, once elicited, persist abnormally, or perseverate and this feature is obvious in both ideational and kinetic apraxia. In magnetic and repellent types of kinetic apraxia the continuation of the reaction can in each case be directly related to continued effect of a natural stimulus. The fundamental types of cortical motor responses, except possibly the optic motor reactions, have a corresponding subcortical mechanism that can operate at thalamic level. For the instinctive grasp reaction the corresponding subcortical response is the tonic grasp reflex, and for the instinctive avoiding reaction the subcortical response is an exaggerated overextension of fingers associated with pronation and flexion of the wrist (tonic withdrawal). These are seen with that type of disturbed attitude we call dystonia. In place of the perseveration of response seen in cortical lesions these subcortical reactions show powerful tonic persistence, actively resisting all contrary movement. Dystonia is more resistant to passive displacement than either spastic or plastic rigidity. Although the basic overresponse to stimulus is still present, intense reaction continues for long periods without apparent stimulus. It is this type of persistcence that gives the response its "tonic" quality.

Thus in dystonic grasping we may see the hand tightly clenched and supinated, closing more tightly with every attempt to open it (Fig. 10, A). The patient is unable to open it to order. The hand can be opened only by stroking the dorsal surface of the knuckles

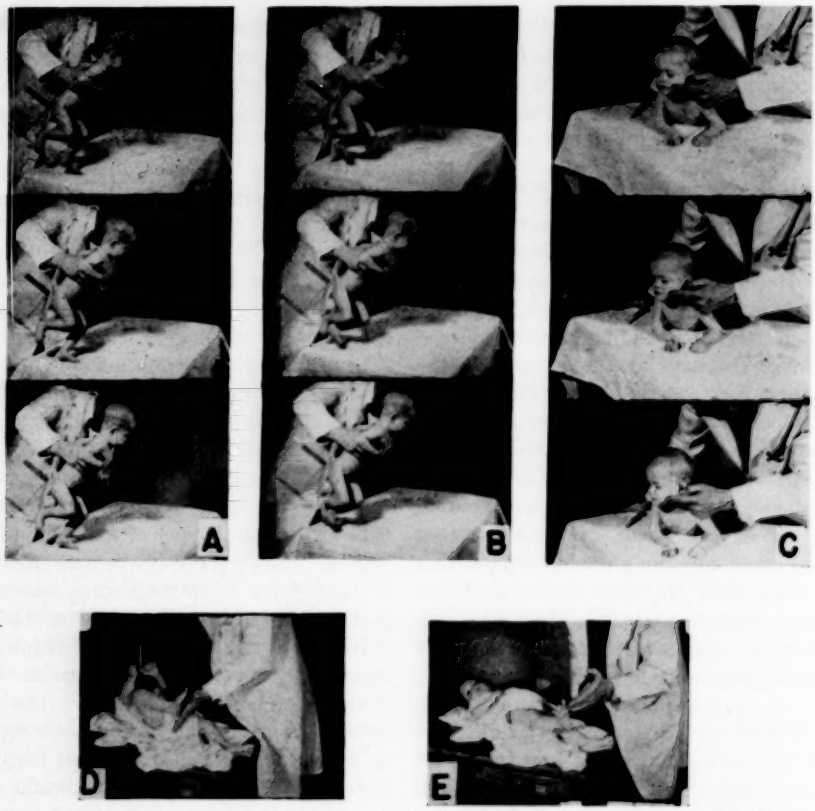

FIG. 9. A, B and C show a child aged one year, with loss of ability to walk or stand following encephalitis, owing to exaggerated avoiding reactions. When lowered to a surface as in A, the feet withdraw immediately contact is made (lowest in A followed by sequence in B). C shows avoiding reaction of head in response to contact with cheek. Avoiding reactions were well developed in all limbs. In D and E strong extension and eventual grasping are shown in the limbs of another child who became stiffly extended whenever propped on the feet.

(thus eliciting a weak and brief contrary avoiding response), or by the presentation of an object to be grasped in the field of vision (Fig. 10, C and D). The woman shown in Figure 10, A once grasped her daughter's hair in her right hand in a fit of rage and was then unable to let go for several minutes. Once open the hand remains open for a few seconds but snaps shut on any tactile stimulus with the palm. In such conditions a combination of a palmar stimulus with a moving contact of the dorsum of the hand leads to rhythmical flexion and extension of the fingers of parkinsonian type. One can thus set up a rhythmic conflict by contrary afferent stimulation. The limb is continuously held in a stiff attitude of partial flexion, increased by any attempt to extend it. This is the "tonic innervation" of Wilson and Walshe (58). The jaw of the same patient commonly exhibits the "bull-dog phenomenon," clenching tightly on any object between the teeth (Fig. 10, B). The eyes once closed cannot be immeditely opened. The foot will exhibit the tonic foot response of Goldstein (21, 22), the toes and sole flexing strongly on any heavy contact with the ball of the foot (Fig. 10, E, F). The foot flexes further

FIG. 10. Dystonic grasping. A. Posture of right hand at rest in a woman with right sided dystonia. B shows the "bull-dog" closure of the jaws when the mouth is stimulated with a towel, in a patient with Wilson's disease. In C the same patient is shown unable to relax the grasp in the dystonic right hand (the left also shows a dystonic grasp). In D the right hand automatically releases when the examiner tells the patient to grasp his other hand. In E the tonic foot response is being elicited in the same patient, persisting after many seconds in F.

when put to the ground. Tonic flexion may persist for long periods without any stimulus, the limb being otherwise stiffly extended. When the condition is uncomplicated, the plantar response is flexor, though painful stimulation may elicit a brief extension of the great toe as part of a defense withdrawal. The feet lag in walking, the body in flexed posture, leaning forwards. There is great difficulty getting up from a chair. This type of movement disorder is seen in relation to lesions encroaching on the anterior thalamic

region and is attributed by us to overaction of the pyramidal system; yet the patient may be unable to use the limbs if the condition is severe. Once marked release of stretch reflexes is caused by extension of the lesion, the tonic grasp and tonic foot response disappear, and are replaced by clonic spasticity.

In contrast we see occasionally another condition of tonic extension and pronation of the hand, with stiff attitude of the limb which is held flexed, particularly at the wrist, as in the left hand of the patient seen in Figure 11, D. The foot is inverted with overextended great toe (a "permanent Babinski") as in Figure 11, A. Any light stroking contact with the foot leads to a further extension of the great toe (Fig. 11, B, C). The lower limb is stiffly extended, but tends to flex when put to the ground. In

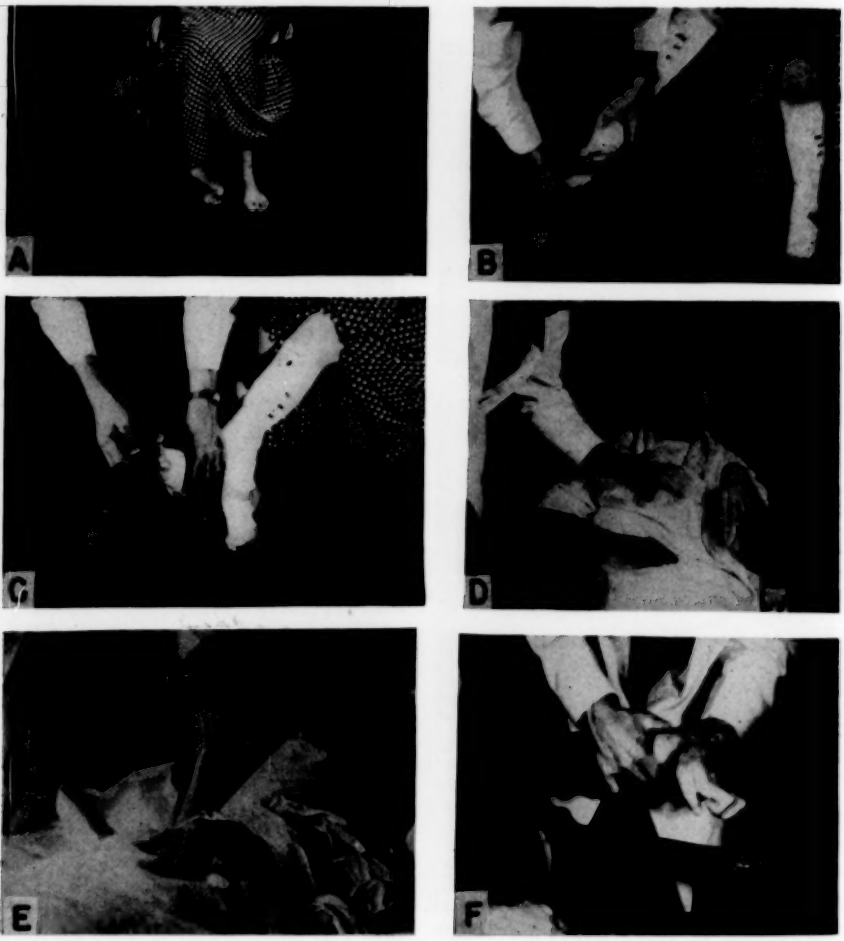

FIG. 11. Dystonic avoiding in the right foot, note overextended toes and inversion of ankle. B shows further extension of toes with plantar stimulation, and C an earlier type of response in the left foot (progressive extrapyramidal disease, ? post-encephalitic). D shows the characteristic attitude of dystonic avoiding in the upper limb (same patient as Fig. 7). E shows the dystonic avoiding posture in the hands in the terminal stage of paralysis agitans. F shows the "thalamic hand" in a patient with hemiplegia and severe thalamic syndrome.

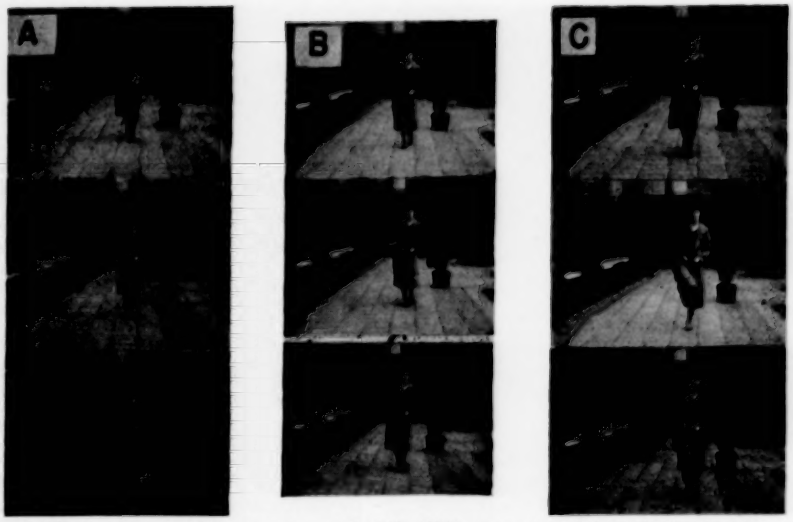

FIG. 12. The "peacock gait" in a post-encephalitic. With every step of the right leg the hip overflexes, raising the leg high from the ground where it is held for about 2 seconds (A, lower) before being extended. The step with the left foot, B, is natural. C shows the overflexion of the right hip with the following step.

early stages progressive flexion of the limb occurs in walking ("peacock gait") (Fig. 12). The mouth is commonly held open and opens further with contact with the lips. The eyes blink seldom. When the patient attempts to grasp an object, the hand seems to hover over it and delays approaching it closely, and the fingers extend further instead of flexing. This condition, exemplified by the left hand in Figure 7, E, is commonly called "athetoid," and if the posture is unstable it constitutes athetosis. In some cases closure of the hand by effort of will is only partial or even impossible but can be facilitated by contact with the palm. In this type of dystonia, commonly seen with lenticular and related lesions, there is overwhelming preponderance of repellent subcortical reflex effect, completely or partially out of control of willed effort. The typical posture of the hand appears in a late stage of paralysis agitans (Fig. 11, E).

In diffuse types of extrapyramidal disorder such as Wilson's disease or the residual states of lethargic encephalitis where dystonic states are prominent, the repellent type of dystonia often affects the mouth and eyes, giving the wide eyed expression with open mouth that is very characteristic of the juvenile type of Wilson's disease (Fig. 13). Since the mandible is actively held open for long periods during a stage of active development, there is then not only drooling of saliva but a downward deformity of the mandible itself. The front teeth will not meet those of the maxilla, even when the back teeth are opposed (Fig. 12, B). Articulation is poorly performed. Pseudobulbar phenomena such as overdisplay of emotion are later additional features independent of the dystonia. In another type of juvenile Wilson's disease magnetic dystonia results, presumably from a different incidence of lesion, as in the patient shown in Figure 10, B to F.

These two disorders, dystonic grasping and dystonic withdrawal, are generally regarded as extrapyramidal postures. Examination of their response to contactual stimulus reveals that they are but coarser subcortical responses interfering with movement in the same sense as what we have called cortical apraxia. The patient is unable to initiate certain movements because the

FIG. 13. Chronic mild dystonic avoiding in a patient aged 15 with Wilson's Disease. Note the pronated posture of the outstretched fingers with slight flexion of the wrists, in A. The almost continuously open mouth and infrequent blinking of the eyes are characteristic. The avoiding lip reaction, B, was regularly elicited. The persistence of this facial dystonia for 2 years resulted in inability to bring the front teeth together when the molars meet, C, owing to resulting deformity of the mandible.

whole motor mechanism is biased in the opposite direction.

"PYRAMIDAL" FUNCTION

The neck and righting reflexes of Magnus and de Kleyn are often spoken of as if they represent a nervous mechanism separate from that of purposive "willed" movement. Such a view is inherent in theories of motor function current in the early years of this century when posture and movement were regarded as two entirely separate types of neural reaction. Yet close observation of the events of hemiplegia in man reveal that the two are inseparable. In a previous paper in this journal we have shown that in the disintegration of motor ability resulting from progressive damage to the precentral region in man, the loss of function proceeds from magnetic apraxia through a phase of reflex grasping to release of the traction reaction, a proprioceptive response that determines the spastic hemiplegic attitude. In recovery the reverse sequence occurs. Twitchell (56)

in his further analysis of a series of cases of hemiplegia showed how recovery progressed systematically through these same stages, and could be arrested at one or other stage if incomplete. He also showed in man, what we had together earlier observed in monkeys, that as each new reflex response appeared in the process of recovery, the individual rapidly learned to adapt the same movement to purposive effort without the natural reflex stimulus. These studies made it clear that what we call willed movement is in fact the learned ability of the organism to utilize movements that are primarily reflex for purposes that involve a more tenuous stimulus situation.

The observations we have reported here do not alter this conception. They indicate only that the modulation of instinctive grasping by instinctive avoiding is necessary for the complete control of movement. If the reflex situation is itself strongly biased, all purposive effort is correspondingly limited because one or other type of reflex response is disproportionate to stimulus. This is the essence of kinetic apraxia.

Following the studies of Tower (55) in 1933 and 1935 it was no longer possible to relate purposive movement solely to the operation of the pyramidal system, or categorize any type of movement as belonging to that system. The common association between paralysis and damage to the pyramidal tract in man certainly points to an important part played by this pathway in movement. Exploratory movement projected in space appears to be particularly dependent on integrity of the pre-central gyrus and pyramid. In its absence some other mechanism leads to dorsiflexion of the great toe in response to moving contact with the sole of the foot and often of extension of the fingers in response to a rapid distally moving contact with the palm of the hand. A special variant of such reaction is that called the Babinski response, elicited with a somewhat heavier contact that gives a more constant and reliable sign. That cortical paralysis can occur without these changes should occasion no surprise, for cases with subsequent verification of integrity of the pyramidal tract have been reported by Spielmeyer (54), Rhein (49), Finkelberg (18), and Bielschowsky (7). The question has been recently reviewed by Lassek (29, 30). The nature of such paralysis has not been analyzed by these authors.

The observations reported here are related in terms of their falling into two groups of consistently associated phenomena. We have studied cases of magnetic apraxia where the medial frontal region was damaged by occlusion of branches of the anterior cerebral artery, or by tumor, but by the time the most completely studied cases came to autopsy the lesion was too extensive for exact anatomic analysis. Similarly though the essential features of repellent apraxia can be found in intraparietal softenings of the type figured in a recent paper by Denny-Brown and Chambers (15), the most fully developed condition has been studied in patients who are still alive or in whom brain tumor has produced only a very extensive lesion by the time the brain can be anatomically examined. Similarly, our best instances of dystonic grasping were suffering from temporosphenoidal ridge tumors, of which it can only be said that the anterior thalamic nuclei and related fiber bundles were early affected. The most uncomplicated dystonic avoiding has been seen in situations where early damage to the lateral thalamic nuclei or putamen were the situaiton of maximum pathology. Exact anatomic study awaits the occurrence of sufficiently well documented cases with strictly localized pathology. Experimental studies in the macaque monkey have shown the general consistency and cortical pattern of these two chief types of kinetic apraxia.

Analysis of dystonic attitudes, and the corresponding postures of athetosis has been confused in the past by their frequent association with undoubted pyramidal disorder. As a result of the common type of capsular lesion, the hemiplegic hand exhibits

either a posture of closed supinated fist, with corresponding partly flexed and inverted foot, or conversely the "thalamic posture" of overextended metacarpal joints, pronated wrists, and everted foot, complicated by clonic spastic flexion of the terminal phalangeal joints (Fig. 11, F). The brisk and clonic reflexes document the pyramidal lesion, but the Babinski sign is facile in the first and difficult to elicit, or absent, in the second type, even if no movement is possible. The rigidity is then a mixture of dystonia and spasticity, the presence of the latter being often shown only by a poorly developed clasp-knife phenomenon and increased tendon reflexes.

Also it should be noted that most commonly extrapyramidal diseases with diffuse pathology, such as Wilson's disease, or lethargic encephalitis, result in asymmetric and incongruous release, for example of avoiding response in one hand and dystonic flexion in the corresponding foot, or conflicts between avoiding and grasping in the same hand. The task of the present communication has been to unravel the essentially congruous reactions.

If, as we maintain, dystonic attitudes represent the overactivity of one or other of two mechanisms for the control of movement that can be independently disordered at thalamic level, how are these related to the Rolandic (pyramidal) motor system? All the available evidence points to the existence of a mechanism for motor reaction at thalamic level for each of the cortical types of motor reaction including Rolandic function. The long cortico-spinal projection system of the pyramidal tract appears to be the chief efferent pathway of a highly specialized type of contactual exploratory response, with subcortical components, both efferent and afferent, in the diencephalon. We therefore distinguish two general types of motor behavior, positive exploratory, with pyramidal and extrapyramidal components, and negative avoidance with extrapyramidal mechanism. Both extrapyramidal components have cortical and subcortical levels of management .Disturbance of any one of these leads to overaction of the others.

The loose flabby fingers with mild proximal spastic traction reaction and large slow tendon reflexes, resulting from hemispherectomy, represent pyramidal release with absence of both magnetic (grasping) and repellent (avoiding) reactions. The intense spasticity that characterizes some capsular lesions is attributed by us to facilitation by concomitant perseveration of grasping or avoiding reactions also released by lesion at that level. The fully developed Babinski sign in cerebral lesions, in the sense of extension of the great toe with fanning of the others, appears to result from release of the avoiding responses at highest level owing to loss of all or part of the exploratory responses. The type of extensor toe response alone elicited by the moderately heavy stroke of a blunt object appears to be a reliable sign of disturbance of the more specialized function of the Rolandic cortex and pyramidal tract. Release of defense withdrawal of the whole limb including dorsiflexion of the great toe is a result of release of the flexion reflex at lower levels.

CONCLUSIONS

We conclude that in its highest elaboration mimetic and propositional behavior is related to conceptual organization of space, objects and persons, in the dominant hemisphere. Such behavior is more highly vulnerable to pathologic changes than is direct physiologic reaction to external events. The resulting disorder is ideational apraxia. Perseveration of identification is a mild form of the same defect. Acquired specialized motor function that requires a leading hand for its performance is closely related to cortical dominance but can be affected independently of symbolic function. The relationship is probably through the conceptual defect underlying constructional apraxia. Related symbolism and dexterity may be separately managed by different hemispheres, inde-

pendently vulnerable to unilateral lesion, and independently controlling the subcortical motor apparatus for the two sides.

Apraxia that is independent of conceptual function (kinetic apraxia) can be traced to the abnormal operation of released sensory factors that direct the physiological aspects of behavior. There are two general types of disturbance, exemplified by frontal and parietal kinetic apraxia, both of which show perseveration of sensory effects. Bilateral kinetic apraxia first and most seriously affects gait and other bilateral motor performance. When the normal equilibrium of cortical praxis is already disturbed, it is possible to induce tremor by concurrent stimuli with conflicting effect. Subcortical extra-pyramidal lesions release the same apractic reactions in persistent (dystonic) form. There are more commonly mixed forms where conflicting reflex effects lead to instability of posture, athetosis and tremor. The static (tonic) aspects of such disorders of movement can be identified with the prolonged operation (perseveration) of sensory factors.

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SOME ASPECTS OF AMINO ACID AND PROTEIN METABOLISM OF THE NERVOUS SYSTEM[1]

HEINRICH WAELSCH, M. D., Ph. D.[2]

The biochemist concerned with the biochemical basis of mental disease has the choice of several avenues of approach, some of which have during recent years come into the foreground of interest. The central nervous system presents to him a collection of different morphologic structures with different metabolic patterns. The interplay of these metabolic patterns, occurring in a variety of structures, and their relation to the biochemistry of the whole organism is at present only superficially understood. One might suspect that abnormalities of such interrelationships are among the causes of mental disease. But it is not possible to evaluate the effect of the metabolism of the whole organism on the central nervous system in health and in disease without a study of the metabolism of this organ system detached from its environment. Only by such a study can we hope to obtain something like a metabolic base line approximating a norm even though fictitious; at the same time we might perhaps get an estimate of the degree to which the central nervous system is an autonomous metabolic unit with its abnormalities residing in its own metabolic processes.

If we want to inquire into the nature of those metabolic activities of the central nervous system, whose abnormalities could contribute to aberrant functioning of the brain, our attention is directed towards the enzymatic processes of the organ system as a whole and particularly of its functional parts. Since the carriers of enzymatic activities are proteins, their metabolism assumes importance beyond that of their being the basic structural elements of the brain. Among their manifold functions, proteins of the central nervous system may be responsible for storage of information, one of the specific tasks of the nervous tissue. If one may be permitted to speculate here, one may assume that information is stored and released in a dynamic way, by a trigger reaction initiating a particular and unique pattern of physico-chemical metabolic changes, or that subtle changes in the configuration of protein macromolecules may represent the imprint of acquired information. Whatever information the biochemical approach may yield, whether it is applied to animals or to man, normal or mentally ill, it must be realized that one can only hope to gather insight into biochemical processes which parallel normal or abnormal functioning of the nervous system without at present fathoming the connection of such processes and the working of the mind.

Our main concern in this presentation will be the metabolism of nitrogenous compounds, in particular of amino acids and proteins in the central nervous system, a subject which has occupied the writer's laboratory for many years. Amino acids are not only the building blocks from which proteins including proteins of hormone nature are made but they also are the mother substances of many active amines, such as noradrenalin, adrenalin, serotonin, and acetylcholine, and the components of neurohormones of peptide nature, such as those of the posterior pituitary. The free amino acids, furthermore, fulfill an impor-

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[2] From the New York State Psychiatric Institute and the Department of Biochemistry, College of Physicians and Surgeons, Columbia University, New York, N. Y.

tant role in the maintenance of the ionic environment of the nerve cells. The pool of free amino acids of the central nervous system is in its composition characteristic for this organ. It contains glutamic acid, in a concentration higher than in any other organ (18, 20), and a high concentration of acetyl aspartic acid (17), the occurrence of which is unique for the nervous system. In addition, it contains a high concentration of γ-aminobutyric acid derived from glutamic acid by enzymatic decarboxylation (14). The amino acids which make up the free amino acid pool may be either synthesized in the brain itself or derived from the circulating blood. The latter mechanism, the uptake from the circulating blood, must operate for those indispensable amino acids which cannot be synthesized by the mammalian organism. The rate of uptake by the brain of amino acids, indispensable or not, is under the control of the blood-brain barrier.

In order to study the role of the blood-orain barrier in amino acid metabolism of the brain, the flux into the brain of two amino acids with opposite physico-chemical properties has been measured in our laboratory (9, 10, 20). One, glutamic acid, is an acidic amino acid, the carbon skeleton of which can be synthesized from glucose through the citric acid cycle; the other is the basic amino acid, lysine, which, as far as we know, cannot be synthesized by the mammalian organism. If we now measure the flux of these amino acids, isotopically labeled, from the circulating blood into the brain, we find a very rapid movement from the blood into the pool of free amino acids of the brain (Table 1). This movement is so rapid that the free lysine of the brain is replaced within one hour by lysine derived from the blood. The movement of glutamic acid from the blood into the brain is somewhat slower (6); since there is so much more glutamic acid than lysine in the brain, and about five times as much lysine than glutamic acid in the blood plasma, it will take very much longer to replace all the glutamic acid of the brain by blood glutamic acid. Although there is rapid exchange of these amino acids between blood and brain, it appears impossible to raise the concentration of these amino acids in the brain substantially by raising their blood concentrations (7, 16). These results suggest that exchange of the amino acids between blood and brain is permitted but that a net increase of the amino acids in the brain is somehow hindered. It is of interest to note that this situation is different in young immature animals, in which the blood-brain barrier apparently does not function fully and permits net increases in the brain of intravenously administered amino acids (4, 7).

TABLE 1

Flux of Glutamic Acid and Lysine from Blood to Brain

	Plasma	Brain	Flux	$t_{\frac{1}{2}}$
	mg. %	*mg. %*	*mg./100 g. tissue/hr.*	*hours*
Glutamic acid	2	150	8–11	8–12
Lysine	6.3	3.7	4–31	0.75

Flux = mg. of amino acid transferred to 100 gm. of brain per hour

$t_{\frac{1}{2}}$ = time in hours needed for replacement of one half of the free glutamic acid or lysine respectively of brain by corresponding amino acid of blood.

We know next to nothing about the mechanisms which regulate the blood-brain barrier or about internal or external toxic substances which may influence it (19); nor do we know whether or not old age means a loosening or tightening of the barrier. We can see clearly that a disturbance in the function of the blood-brain barrier must have an effect on the metabolism of the central nervous system. Such disturbances could express themselves in local or general excess or deficiency of metabolites, and in the case of amino acids, in changes of their availability for protein synthesis, for the production of active amines, and for the maintenance of the ionic environment.

The observation that free exchange of amino acids between blood and brain may be permitted while net increase of amino acid concentration in the brain is hindered, raises the question of whether or not the systems responsible for the transfer of amino acids are controlled by the concentration of amino acids within the brain. One might ask, therefore, whether the blood-brain barrier would act in a similar manner towards metabolites not present normally in this organ, a problem which has not been approached as yet.

Amino acids represent the building blocks of the proteins to which we shall direct our attention now. It seems of interest to inquire first of all into the overall metabolism of the brain proteins, *i.e.*, the speed of their replacement. As pointed out above, the proteins are not only structural elements and carriers of enzymatic activities, but may also be the macromolecules representing the basic units for the storage of information. Furthermore, gene-determined abnormalities of the function of the nervous system might find their material basis in the complete absence or the rearrangement of specific proteins with increase or decrease of the content of specific amino acids.

The solution of the problem of the rate of replacement or turnover of proteins of the central nervous system has been hampered by the lack of methodologic approach—a fact which has led to the extreme assumption that the proteins of the brain do not turn over at all, and to the denial of the dynamic state of brain proteins. This notion was made unlikely by the indirect observations of Hyden (5) some years ago but was definitely disposed of by the results of studies carried out by Gaitonde and Richter (2) and in our own laboratory (8, 9, 10, 20). The findings in both laboratories may be summarized by the statement that the proteins of the central nervous system have a rapid turnover which is not much below that of the liver proteins. Our experiments were carried out with the aid of isotopic lysine. It was found that by varying the time period between intravenous administration of the amino acid and sacrifice of the animals, it was possible to measure different protein fractions with differing turnover rates in the experimental procedure. Thus, a whole spectrum of turnover rates could be observed (Table 2). The fastest protein fraction in total mouse brain has a turnover corresponding to a half-life time of three days while in the same experiment, the fastest protein fraction of liver had a half-life time of one day. Without going into detail, suffice it to say that in long time experiments lasting 60 minutes, protein fractions with a half-life time of 15 and more days were found. These results underscore the fact that the proteins of the brain, as of any other organ, are heterogeneous in their turnover rates. The question now arises whether the different functional areas of the brain have proteins with a half-life time which may extend the spectrum of turnover rates even further. It can be shown easily that the different functional areas of the brain contain proteins with different turnover rates (10, 11). In our experiments with monkey brain, we were surprised to find that the corpus callosum and its radiation contained the proteins with the highest rate of turnover, followed by the proteins of the cortex and cerebellum, thalamus, hypothalamus, pons, and finally of the cord (Table 3). When the cells of the cortex of the monkey brain were fractionated into nuclei, mitochondria, microsomes, and soluble proteins, the microsomes showed by far the highest rate of turnover of isotopic

TABLE 2

Half-life Time (Days) of Proteins of Mouse Organs

Time, after injection of C^{14}-Lysine	Brain	Liver	Muscle
minutes			
2	2.8	0.9	3.5
5	3.5	1.3	7.6
10	5.5	2.6	7.6
30	6.9		13.2
60	15.2		23.6

TABLE 3

Half-life Times of Proteins in Monkey Brain Areas

	Half-life time in days calculated from different time points		
	5 min.	10 min.	45 min.
Cord	10.6	9.5	18.5
White	3.9	4.0	8.7
Medulla-pons	6.6	6.7	13.7
Thalamus-hypothalamus	6.1	7.8	16.8
Cerebellum	5.4	7.6	14.1
Cerebral cortex	5.5	6.7	12.7

TABLE 4

Incorporation of C^{14}-Lysine into the Proteins of Different Cell Fractions of Brain Cortex (M. irus)

(30 minutes after injection)

Composition of Fraction	Counts/mg. Lysine/min.
Cell debris, whole cells, nuclei, large mitochondria	500
Mitochondria	400
Microsomes	800–1400
Supernatant fluid	600

lysine. The rate of incorporation into microsomes was three to five times the average value for the total cortex proteins (Table 4). One may, therefore, suspect that some proteins of the brain cortex, *e.g.*, those of the microsomes, turn over with a half-life time of 15 hours or even less, since these fractions also are in themselves not homogeneous but mixtures of fast and slowly metabolizing proteins. It is, of course, of particular interest that the microsomal proteins of the brain show such a rapid turnover since modern electron microscopy has equated the microsomes of the cortex with Nissl granules (13). Our findings are in keeping with the observations of rapid changes in the appearance of Nissl granules in situations of stress.

The overall picture of the metabolism of the proteins of the brain suggests that their rapid metabolism may contribute to some of the functional properties of this organ beyond their being structural components and participating in energy metabolism. It has been reported above that the proteins of the corpus callosum showed a higher rate of turnover than did the proteins of other areas of the monkey brain. Since the results are based on the rate of incorporation of C^{14}-lysine into the proteins of the corpus callosum and do not differentiate between protein of the axoplasm and the glia, the possibility cannot be excluded that it is the proteins of the glia in this area that show a particularly high rate of turnover. Furthermore, in the calculation of turnover rates the specific activity of the protein lysine is related to the specific activity of free lysine in the pool of free amino acids of this area; thus, a low specific activity of lysine, together with an average specific activity of protein lysine would result in the calculation of a high turnover rate. It is by no means excluded that, in the calculation of the turnover rate of the proteins of the corpus callosum, the average value of free lysine found in this area does not actually represent the lysine compartment from which the amino acid for protein synthesis is derived. One does not need to emphasize the importance of reference to the proper compartment of precursors in all calculations of turnover rate.

But irrespective of what may be the explanation for the high turnover rate of the proteins of the corpus callosum, the above observation served to direct our attention to protein synthesis in structures with axons as the main elements. Therefore, the incorporation of C^{14}-lysine into different segments of the sciatic nerve of the frog and of the spider monkey was studied (12). In experiments lasting up to 50 days the specific activities of the protein lysine of the sciatic plexus and sciatic nerve of frogs were after five days highest at the proximal segment of the plexus and lowest at the distal end of the nerve (Table 5). As the time interval between administration of isotopic lysine and sacrifice of the animals increased, the specific activities of the proxi-

mal nerve portion decreased considerably while those in the distal portion rose slightly. Whereas similar results were obtained in a number of experiments with frogs, the incorporation of radioactive lysine into the sciatic nerve of the spider monkey failed to show such a time dependent trend. The results of the experiments carried out with frogs might suggest the possibility of a flow of axoplasm through the nerve, the proteins being synthesized in the cell body and migrating down the nerve. Such an intriguing possibility of flow of axoplasm has been discussed by Weiss and Hiscoe (22) and Gerard and his associates (3); however, at present other interpretations may offer an explanation for our experimental results. It is, for example, obvious that a decreasing rate of turnover of the nerve proteins along the nerve in proximo-distal direction would give the same picture as would a gradient of C^{14}-lysine flow through the nerve. However, it seems at present unlikely, in view of electron microscopic evidence, that the axoplasm contains the system responsible for protein synthesis. A further unknown in the interpretation of results on protein synthesis obtained with myelinated nerve is the extent to which the metabolism of Schwann cells contributes to the overall picture. Nevertheless, the determination of the origin of nerve axoplasm protein is of major significance not only as a possible clue to the origin of neurohormones of peptide nature; it also has important bearings on the problem of pharmacologic effects on the nerve through influence on local protein synthesis or flow of axoplasm.

We have up to now been concerned with the overall metabolism of brain proteins. I should now like to discuss a particular protein fraction with a new, probably enzymatic, activity, recently discovered in our laboratory, which may be of significance for the metabolism and function of biogenetic amines. When purified protein fractions of various organs were incubated with one of a variety of C^{14}-amines such as cadaverine, putrescine, histamine, etc., the amine became and remained attached to the protein even after treatment by various procedures designed to remove adsorbed C^{14}-amines or amino acids (1, 15). This incorporation was most marked in liver, less in kidney and least in brain. Two relatively simple mechanisms suggest themselves as possible explanations for the amine incorporation reaction. First of all, the amino group of the amine might exchange with the amide group of protein bound glutamine or asparagine, a reaction which would lead to the formation of proteins containing the amine bound in amide linkage. An alternate possibility would be a transpeptidation reaction by which the amine exchanges with an amino acid or peptide by aminolysis of the peptide chain. These are the two mechanisms which at present serve as working hypotheses in our laboratory for an explanation of the incorporation of amines into the active protein fraction. The incorporation of amines into the protein fraction does not seem to be restricted to any particular C^{14}-labeled amines and includes $N^{15}H_3$. When it was found that serotonin also was incorporated into the purified protein fraction of guinea pig liver, the question arose whether this amine incorporation reaction might perhaps have specific significance in the nervous system. In liver, the active fraction was prepared from the soluble protein fraction after removal of nuclei, mitochondria and microsomes by fractional centrifugation. Both cadaverine and serotonin were incorporated

TABLE 5

Lysine C^{14} Incorporation into Sciatic Nerve Proteins

(Rana p.)

Cts/Min./100γ Lysine Upper Portion Plexus = 100

Days	Liver	Cord	Sciatic Plexus		Sciatic Nerve	
			Upper	Lower	Upper	Lower
8	220		100	51	28	25
17	150		100	73	29	35
28	260	130	100	83	63	63
38	300	170	100	100	100	116

about equally well into this liver fraction. The corresponding fraction of brain showed only slight and often doubtful incorporation of serotonin or any other amine. An investigation of the incorporation into the different cellular fractions of brain showed extensive incorporation of serotonin but not of cadaverine into the proteins of brain mitochondria (21). It should be emphasized that at present the mode of linkage of the incorporated amines is still unknown, and that equally little is known about the mechanism of serotonin incorporation. Further studies will show whether this amine remains unchanged and becomes attached to proteins, or possibly other constituents of the mitochondria, in a manner similar to the attachment of other amines to the soluble protein fraction of the liver.

It is of course not possible at present to draw definite conclusions as to the biologic significance of this amine incorporation reaction; nevertheless, it is apparent that, if occurring *in vivo*, it may lead to the formation of modified proteins which in turn may affect permeability, enzyme activity and immunological reactions. These observations also open up the field of inquiry into the action and function of protein bound amines, a problem of particular interest with respect to agents such as serotonin or histamine. If a mechanism of amine incorporation by exchange with amide groups were found to be operative, it would be of significance for the ammonia metabolism of brain and other tissues. If, on the other hand, aminolysis of the peptide chain were found to occur, it would be interesting to see if peptides are liberated and have any physiologic function.

GENERAL CONSIDERATIONS

In this short summary, based mainly on work carried out in the author's laboratory, some general aspects of protein metabolism of the nervous system and of the effect of the blood-brain barrier on amino acid uptake by the brain have been presented. Furthermore, the possible significance of a new amine incorporating system occurring in brain and other organs has been discussed. It is undoubtedly tempting to make these findings, together with the significant contributions of other laboratories to this area of neurochemistry, a basis for speculating on interpretation and etiologic factors of mental disease. However, in no other field of fundamental medical research is the investigator under stronger obligation not to extend his speculations beyond the confines of a working hypothesis open to test by feasible experiments.

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VISCERAL BRAIN, ITS COMPONENT PARTS AND THEIR CONNECTIONS[1]

JAMES W. PAPEZ, M.D.[2]

The visceral brain or rhinencephalon consists of several closely connected mechanisms, alerting, sensory, visceral, emotional and autonomic. These parts include olfactory, habenular, visceral, amygdalar, hippocampal, hypothalamic and pituitary structures. They are closely integrated to coordinate sensory events with visceral and bodily reactions and visceral needs. They are biologically grounded, closely bound in action to regulate innate, automatized activities concerned with searching, feeding, sex, fight, flight and the emotion provoking situations in the body and in the environment.

OLFACTORY SYSTEM

The olfactory apparatus is ancient in origin and is the basic tubular structure from which other portions of the visceral brain are elaborated with the exception of the hypothalamus. In fishes and the amphibians, the olfactory bulbs are formed from the terminal part of the forebrain vesicles, which are reached by the olfactory nerves coming from the olfactory hair cells in the olfactory sacs. The olfactory bulbs are connected by nerve fibers to the walls of the forebrain vesicles where rudiments of the olfactory and preoptic areas, amygdala (archistriatum), basal ganglia, fascia dentata and hippocampus are developed. Among the earliest connections are those to the habenula and reticular formation, to the hypothalamus and to the hippocampal rudiment (11).

In lower mammals there is an elaboration of the visceral forebrain in all its parts. The olfactory structures in most lower orders of the mammals attain large macrosmatic dimensions. The visceral cortex, claustrum, amygdala, septum, hippocampal formation and olfactory striatum are also prominent features of brains of macrosmatic mammals. Collectively they form the archipallial or visceral brain. Development of neopallial cortex was a later event which brought with it connections to the hippocampal gyrus.

Olfactory structures on the ventral side of the human brain consist of the olfactory nerves which enter the olfactory bulbs (Fig. 1, OB). The bulbs are connected to the anterior olfactory nucleus and trigone by nerve fibers of the olfactory tracts. The lateral olfactory tract, LT, turns laterally in the cortex that borders the anterior perforate substance. The tract curves to enter the surface of the uncinate cortex, UC, where it ends in the lateral olfactory nucleus of cortical cells of uncinate cortex over the amygdala (Fig. 5, LO). The olfactory sensations arise in this region. The intermediate fibers enter the olfactory tubercle (Fig. 2, OT). The medial olfactory tract (Fig. 1, MO) passes deep to the cortex and ends in the region of subcallosal gyrus, GS. This is the region where the medial bundle of the forebrain MF takes its origin and passes back into the lateral portion of the hypothalamus for visceral responses. Note the extensive connections of the bundle with the islands in Figure 2. The superficial, diagonal band of Broca, D, extends from the region of the lateral olfactory nucleus across the anterior perforated substance in front of the optic tract and ends in the medial parolfactory cortex, GS. It affects correlation between the lateral and medial areas. It lies ventral to the olfactohabenular tract, OH.

The olfactory tubercle, OT, and the underlying islands of cells related to the striatum as seen in Figure 2 are the chief areas for sorting and relaying olfactory impulses to

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[2] Laboratory for Biological Research, Columbus State Hospital, Columbus, Ohio.

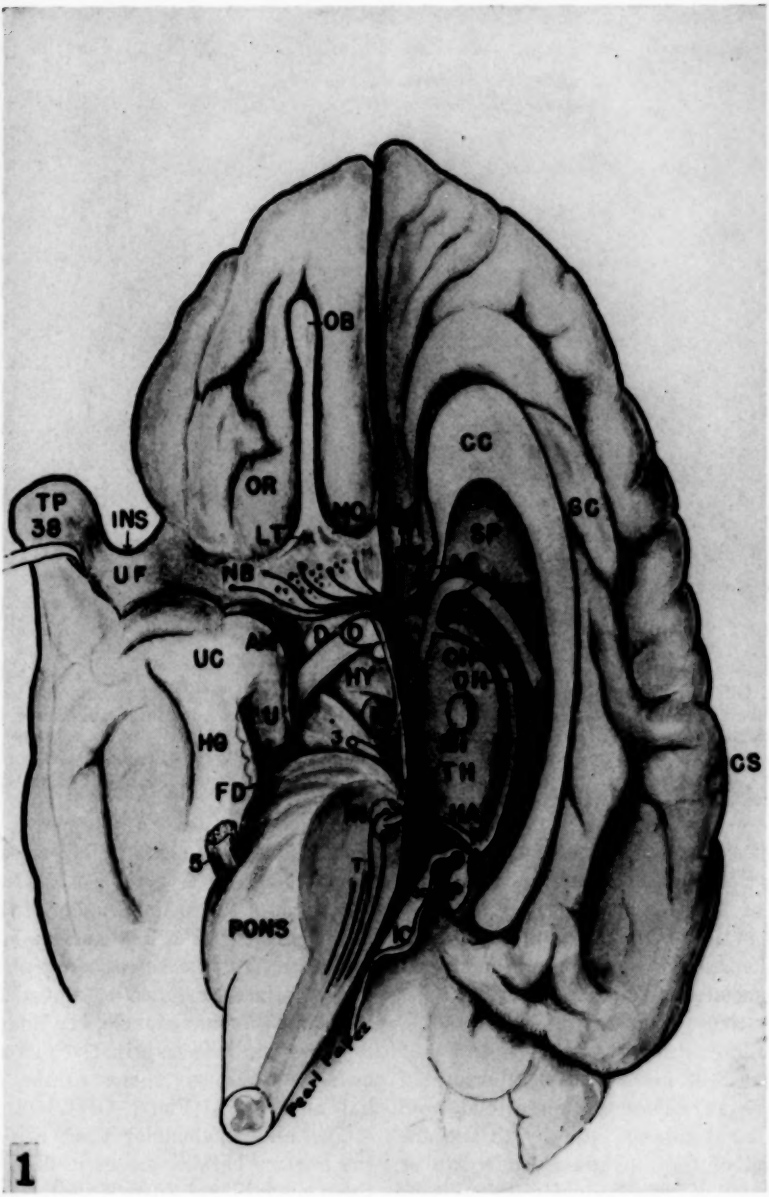

FIG. 1. Olfactory structures in surface of right human hemisphere. Ventral surface seen on left, medial on right. Shows olfactory bulb, tubercle, anterior perforated substance and nucleus basalis, NB. Black lines show origin and course of olfactohabenular and corticohabenular tracts to habenular nuclei, habenulointerpeduncular tract to interpeduncular nuclei, IN, thence to tegmentum, T, and reticular formation. Description in text. $\times\frac{3}{4}$. (See explanation of lettering on pages 55 and 56.)

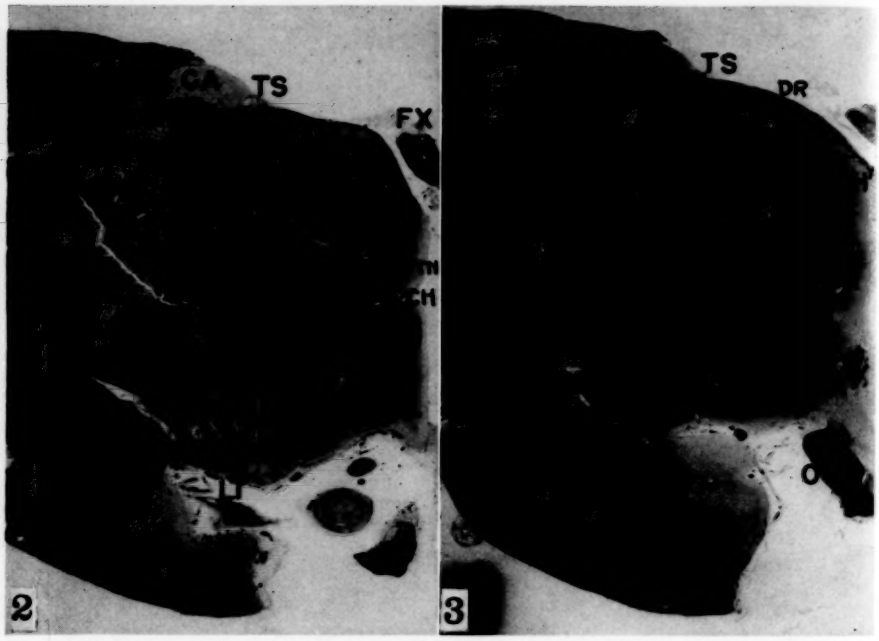

FIG. 2. Olfactory structures, as seen in tubercle. Section through left basal ganglia and level of olfactory tubercle, OT, showing islands of cells surrounded by fibers of medial forebrain bundle, MF, also lateral olfactory tract, LT. Description in text. ×3.

FIG. 3. Visceral structures as seen in frontal section, back of Figure 2, through level of anterior perforated substance, nucleus basalis NB, amygdalar tract, AT, insula, claustral digitations, CL. Note the two divisions of the uncinate fasciculus, UF. ×3.

the nucleus basalis (Figs. 1 and 9, NB) and into the hypothalamus via the medial bundle of the forebrain, MF. Primitive search is in the beginning a function of smell for the detection of sapid, protein foods and other associated reactions.

The nucleus basalis (Figs. 1, 2, and 9, NB) is a long, transversely placed group of multipolar cells in the anterior perforated substance (14). These groups extend from the gyrus subcallosus laterally to the anterior end of the amygdala where two or more groups of cells lie under the anterior commissure, AC. These groups are embedded in the amygdalar tract (Fig. 3, AT). The longest group is posterior to the islands of the olfactory tubercle (Fig. 2, OT). The medial group is embedded in the inferior thalamic peduncle, and is often called nucleus of ansa peduncularis. The most medial group is near the gyrus subcallosus near the origin of the medial bundle of the forebrain (Fig. 1, MF). The nucleus basalis is innervated from the three sources mentioned above, olfactory tubercle, amygdala, and inferior thalamic peduncle, IP. The nucleus may be regarded as a primitive motor center or area which gives rise to the olfactohabenular tract (Fig. 1, OH).

The olfactohabenular tract arises from the nucleus basalis, passes medially above the diagonal band, turns dorsally lateral to the pillar of the fornix (Fig. 1, OH). It ends on the large cells of the lateral habenular nucleus. It is joined by the corticohabenular tract, CH, which arises in the uncinate cortex, passes in the fornix, and leaves the fornix (under the intraventricular foramen,

I) to join the olfactohabenular tract. It ends on the small cells of the medial habenular nucleus. Together the two tracts form the medullary stria of the thalamus, TH. The medial tract consists of fine myelinated fibers and is related to the parataenial nucleus (Figs. 2 and 8, TN). Both tracts have a primitive subependymal position as does the terminal stria, TS.

Habenulointerpeduncular tract [retroflex bundle of Meynert (14)] is also double consisting of myelinated fibers from the lateral habenular nucleus and fine unmyelinated (brown) fibers coming from the medial habenular nucleus (Fig. 1). Both tracts end in the complex interpeduncular nuclei, IN. These in turn give rise to fibers which pass along the tegmental raphé and end about cell groups in that region and in the reticular formation, T. The central and linear nuclei along the raphé seem to be involved. These midline reticular nuclei form a true centrencephalic system (36) (Fig. 10, CE, LI). This ancient "motor" pathway from the nucleus basalis to the bulbar reticular formation and nuclei of the raphé may subserve innate olfactory, visceral, feeding and autonomic reactions. They appear to reach the reticulospinal tracts (24, 25).

This path has complex reticular connections which probably control reactions in primitive search, feeding, defense, etc. Thus sniffing, snarling, nursing, feeding, etc. may accompany and guide muzzle activity. Its relation to grand mal seizures is suggested (36). A tumor on the surface of the medullary stria may cause diencephalic autonomic epilepsy (34). A tumor in the olfactory tubercle may be associated with olfactory hallucinations as well as grand mal epilepsy (27). Olfactory hallucinations bring out the alerted, obsessive olfactory drives and discriminations which otherwise are not exhibited by normal persons. Septal nuclei are probably concerned.

Septal nuclei are rather large structures in macrosmatic mammals. Afferent fibers to them are said to come from the hippocampus by way of fornix. The cells of the septal nuclei appear to send their fibers ventrally and forward to the olfactory cortex (21). Likewise fibers from the anterior thalamic nucleus and frontal end of the reticular nucleus pass to the same cortical region (31, 32). The septal and other fibers probably alert the olfactory cortex, as when an animal follows a scent, sniffs and searches to recognize foods, situations, friends, enemies, etc. (4, 21). Thus smell may be considered as a sense of primitive search, drive and identification of odorous, environmental objects, family members and sanitary conditions (3). This may be related to electrical self-stimulation (4). Septal nuclei send fibers in the medial longitudinal stria LS to the fascia dentata FD and thus control hippocampal reactions (Figs. 6, 7, LS).

Septal nuclei in the primates SP are rudimentary and the basic olfactory drives are not evident as in the lower mammals (4, 21). This is due to the dominance of the visual, auditory, and somesthetic senses employed in primates behavior. Esthetic and foul smells still play a role in many human reactions to perfumes, food and unsanitary conditions. Actually the striatal islands above the olfactory tubercle are numerous and prominent in the human brain (Figs. 2, OT, and 3, NB). The septum is reduced.

VISCERAL SYSTEM

Gustatory and other alimentary functions of taste, thirst, hunger, etc. are developed in the lateral, basal side of the forebrain (35, 37). They are related to the origin of the lateral forebrain bundle which in the course of evolution became the internal and external capsules. Associated oral and bodily reactions are involved in feeding, etc. Here also are developed the basal ganglia with their striatum, and their pallidal divisions connected to the subthalamic and rubral nuclei, important as parts which control muscular tonus, head and bodily movements. These provide another dominant,

route for motor responses aside from the nucleus basalis and medullary stria.

The alimentary system is represented in the visceral cortex of the insula and adjacent orbital cortex (Figs. 1, 2, 3, OR, INS, CL) according to Penfield and Rasmussen (35) and Penfield and Faulk (37). This cortex rises into the subcentral operculum where the acts of swallowing and mastication are represented in the pyramidal system. Abdominal and epigastric sensations, disagreeable taste, nausea, sick feelings and epigastric aura have been elicited by stimulation of these areas and tip of temporal lobe. This alimentary cortex receives fibers from the external capsule (Figs. 2, 3, 4, EX). Inhibitory control of respiration follows stimulation of the visceral cortex which borders the insula (12). The aura and dream states of uncinate fits often include episodes of taste, smell, environment dealing with food, and epigastric aura preceding the convulsive attack. Sucking, chewing, snarling and biting have also been observed following stimulation in ventral locations (41).

FIG. 4. Amygdala as seen in frontal section back of Figure 3, through its front half. Note the fiber bundles around amygdala, UF and AS. Description in text. ×3.

Visceral sensory ascending pathways take their origin largely from the vagal complex of nerve centers of termination in the medulla oblongata (31, 32, 36). The sensory components of the intermediate nerves are included. Fibers from these bulbar centers ascend in the reticular formation mostly in its dorsal, central part. They are present in the primitive vertebrates. Some fibers pass along the dorsal longitudinal fasciculus and may be in contact with dendrites from the central gray matter. In the midbrain the large reticulothalamic tract probably carries visceral afferent fibers to the intralaminar nuclei while some curve ventrally to enter the subthalamus and the region of the subfascicular and submedial nuclei of the thalamus (31, 32). Visceral and reticulothalamic tracts had a common ancient origin. Fibers from the ventral medial nucleus of thalamus terminates in the adjacent orbital cortex. The adjacent ventral anterior nucleus sends fibers forward in the anterior thalamic radiation. These fibers curve over the dorsal margin of the putamen, enter the external capsule and are distributed to the claustrum as well as the visceral (insular) cortex (Figs. 2, 3, 4, EX). The foregoing tracts provide the plausible and objective afferent connections to the visceral cortex and the claustrum.

The claustrum is a deep layer vi of the visceral cortex (Figs. 2, 3, 4, CL). It underlies the insula, lateral orbital cortex and part of the subcentral apercula cortex. Its functions are visceral. Here the presence of callosal fibers indicates a bilateral coordination of oral-visceral functions. The locations of taste qualities, thirst and hunger are not definitely established but are presumed to be in the visceral region (37). Digitations of the claustrum are seen in front of the amygdala (Fig. 3, CL), where they come in intimate contact.

The uncinate fasciculus arises from the

visceral cortex and claustrum, UF. It passes back in the claustrum, divides into a medial part closely associated with the claustral digitations and lateral amygdalar nucleus (Figs. 3 and 4, CL). Its lateral division turns down and ends in the uncinate cortex and temporal polar area 38.

The amygdala is a special mass of nerve cells enclosed in the uncinate cortex from a portion of which it is derived (Figs. 4 and 5, AM). Its nuclear masses may be considered as special devices for bringing organized reactions to the "motor" olfactory nucleus basalis above it and to the hypothalamus via the terminal stria, TS. The amygdala consists of several cellular masses called medial, cortical, accessory basal, basal, lateral, central and the bed nuclei of the terminal stria and amygdalar tract (Fig. 5). The uncinate cortex is the primitive forerunner of the hippocampal cortex. It encloses the oval amygdala. This cortex receives in its medial border the lateral olfactory tract LO. This cortex is connected to the surface of the amygdala by a wide association band, as, which enters its lateral

FIG. 5. Amygdalar nuclei as seen in photograph of frontal section through middle of left human amygdala showing its masses of nerve cells. ×6. AB-Accessory basal nucleus. AC-Anterior commissure. AS-Association tract to amygdala from uncinate cortex. NB-Nucleus basalis above label. B-Basal nucleus of amygdala. BN-Bed nucleus of terminal stria. C-Central bed nucleus of amygdalar tract. CL-Claustral layer vi. i, ii, iii, iv, v, vi layers of nerve cells in uncinate cortex. L-Lateral nucleus. LO-Lateral olfactory nucleus. TV-Lateral ventricular, temporal horn between amygdala and hippocampal uncus. UC-Uncinate cortex.

nucleus. The uncinate fasciculus connects the claustrum to the amygdala.

The nuclei of the amygdala are connected in two ways. The basal and accessory basal nuclei are connected by the amygdalar tract AT with the lateral part of the nucleus basalis in the anterior perforated substance. Probably this is a motor outlet for implementing head, oral and accessory activities (9, 44). The motor behavior and flight-fight pattern may pass by this path. The convulsions induced by electric shock therapy may also take this path. The terminal stria, TS, which consists of five bands of fibers arises from the lateral amygdalar nucleus, passes in the groove between caudate nucleus and thalamus forward, curves down into the preoptic area where its bands terminate among the preoptic granule cells beside the supraoptic, paraventricular and other hypothalamic nuclei (Figs. 8 and 9) (9). It is accompanied by a bed-nucleus of granule cells (30). Probably this is an inhibitor path for control of granule cells and autonomic structures (12). It is suggested that docility, affection, sexual acts, olfactory selection, emotional controls, erotic behavior and abnormal uncinate fits with grand mal seizures and patterns of feeding functions may be organized in the amygdala (41, 44). Its commissural fibers come from its lateral nucleus.

The uncinate cortex (Fig. 6, UC) which surrounds the amygdala is connected to the temporal polar area by a bridge of special cortex, the anterior temporal nucleus, along which such field functions and abnormal ones may be channeled into area 38 and spread into temporal gyri; or abnormal conditions in the temporal polar cortex may be channelled into the amygdala and then by the olfactohabenular connections carried

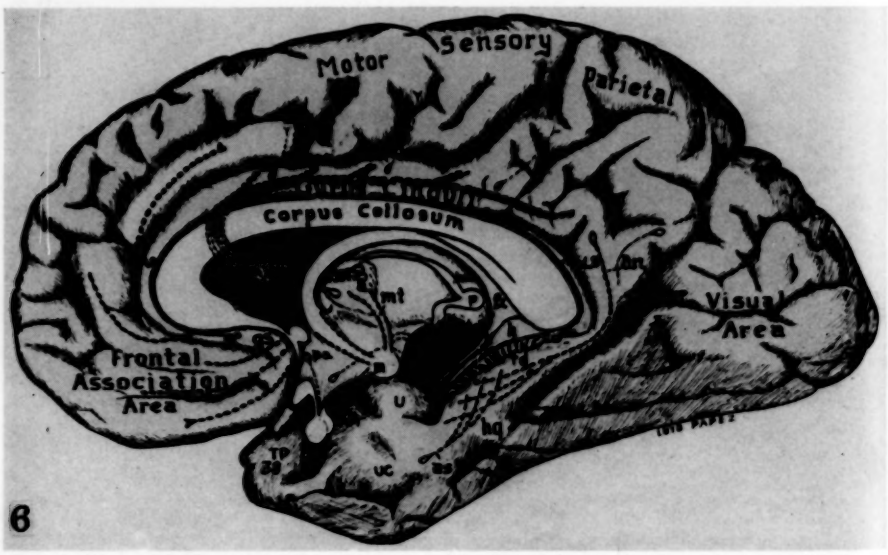

FIG. 6. Hippocampal system as seen in medial view of right human hemisphere showing location of hippocampal formation in medial border of temporal lobe. $\times \frac{2}{3}$. It shows connections of surrounding cortex to hippocampal gyrus, HG, continuity of gyrus with uncinate cortex, UC, position of amygdala, AM, hippocampal uncus, U, fascia dentata, FD, medial longitudinal stria, LS, to fascia dentata, hippocampus, H, on ventricular surface, fornix, FX, going to the mamillary body, M, and mamillothalamic tract, MT, to anterior thalamic nucleus, A, thence to gyrus cinguli. These together represent the hippocampal-cingulate circuit.

down to the reticular formation producing abnormal activity. Another bridge connects the uncinate cortex with the hippocampal gyrus (Fig. 6).

The temporal polar area 38 receives the lateral division of the uncinate fasciculus coming from the visceral areas. We can surmise that various cortical field processes may be represented in area 38, related to food, alimentary functions, appetites, bad sanitary situations, dream states, delusions of poisoned food, aversions to and selection of certain foods, etc. Since the temporal gyri join area 38 such abnormal experiences as tinnitus, vertigo, humming, melodies, visual and auditory hallucinations, repetition of words, and verbigeration may stem from these associated fields. The temporal polar cortex, area 38, also serves to U-turn the field activity back into the four main temporo-occipital gyri.

HIPPOCAMPAL SYSTEM

The hippocampal formation is located in the medial and ventral border of the temporal lobe where it partly encircles the posterior surface of the thalamus. It consists of the hippocampal gyrus, hippocampus, fascia dentata, medial longitudinal stria and fornix (Fig. 6, HG, FD, H).

The hippocampal gyrus, HG, has six layers of nerve cells reached by radial bundles from the underlying white matter (Fig. 7, HG). The fibers which come in impinge on cell groups in layers II and V. The fibers come from several areas of the neopallial cortex, cingulate, parietal, visual, temporal and temporal polar. They converge on the hippocampal gyrus from all directions as seen in Figure 6. The posterior cingulum, AN, comes from the cingulate and precuneal cortex. It forms an angular bundle in the inner wall of the temporal horn of the lateral ventricle (Fig. 7, AN). This bundle brings in impressions from the perineal region and lower parts of the trunk. Fibers from the cuneus and lingual gyrus bring in impressions from the visual area. Several cortical connections from the inferior temporal cortex regularly join the hippocampal gyrus in its middle, often interrupting the collateral fissure. A constant connection occurs between the temporal cortex and the uncinate cortex which contains the amygdala (Fig. 6, AM). No direct sensory afferents to the hippocampal gyrus are known. The primitive uncinate cortex, UC, is a frontal continuation of the hippocampal gyrus. Its olfactory, amygdalar and temporal polar connections are described above. See description under Figure 7.

From the connections listed above it is obvious that the hippocampal gyrus receives transcortical impressions from all surrounding areas of the brain, parietal, visual, temporal, auditory, uncinate, olfactory and visceral. For these it probably forms a common field for a variety of reactions expressing emotional moods and states such as likes and dislikes, love and hate, acceptance and rejection, security and fear, snarling and smiling, attitudes of fight, flight, sex, etc. (7, 13, 16, 18, 19, 26, 41). A strong band of fibers, the outer band of Baillarger is present in the hippocampal gyrus (Fig. 7, AS). This band completes a close association between all parts of this cortical area and suggests that transcortical, field processes whatever their source, perhaps several at the same time, are integrated here and modified to form some of the emotional moods and experiences (26). Removal of the neocortex markedly diminishes these responses (2).

Efferent perforating fibers, PF, arise from numerous and closely packed pyramidal cells of layer v, and angular bundle. They perforate the cortex and enter the subicular stratum in the outer layer facing the hippocampal fissure, SB. They enter this fissure and form a layer of synaptic contacts with the apical dendrites of the hippocampal pyramids. The fascia dentata lies on the outside of this layer.

The hippocampus is formed of large pyramidal cells of a motor type (Fig. 7, H). They are representatives of layer v. The

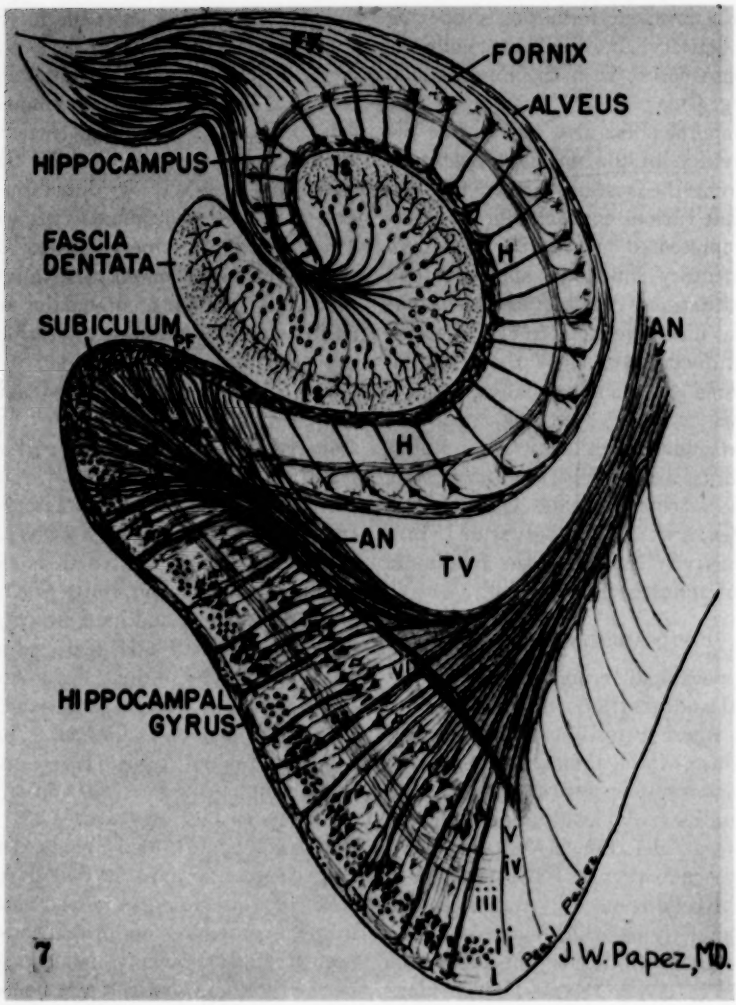

FIG. 7. Hippocampal mechanism. An outline of the cellular mechanisms which are believed to integrate emotional experiences at the conscious level in layers i, ii, and iii, and reactions in dense layer v which has probably the main emissive cells that send out perforating fibers, PF, which contact the apical dendrites of the hippocampal pyramids, H. Medial longitudinal stria, LS, in outer layer of fascia dentata, FD, represents the contacts with dendrites of its granule cells. The axons of these cells terminate around hippocampal pyramids and probably facilitate and inhibit their discharges. AN-Angular, afferent, bundle. Layers ii and v of the hippocampal gyrus are distinctive.

transition from the six layers of cortex of hippocampal gyrus to the single layer of hippocampal pyramid is gradual. These pyramids resemble trees with top branches which radiate into the outer layer where they make contacts with the perforating fibers (Fig. 7, PF). The cell bodies have 3 to 5 root dendrites which branch toward the deep surface close to the alveus. The cell bodies and basal dendrites are surrounded by neurophil and fine axons which come from the granule cells of the fascia dentata. Evolution of the septal and hippocampal formations was described by

Johnston (11). Details of structure are given in Kraft's (1955) translation of Cajal (14, 15, 17, 24).

Fascia dentata, FD, consists of granule cells which represent a primitive layer ii. Their fine dendrites branch toward the surface and make contacts with numerous nerve fibers of the midial longitudinal stria (Figs. 6 and 7, LS). Together they form the outer layer. The origin of the afferent fibers seems to be from the medial subcallosal and septal cortex. This origin is seen in amphibians and reptiles where the corpus callosum is not present (11). In mammals the fibers perforate or circumvent the rostral part of the corpus callosum, gain its dorsal surface as the medial longitudinal stria, pass back of the splenium and enter the outer layer of the fascia dentata. The granule cells probably facilitate and control the firing action of the hippocampal pyramids. Fluid spaces between the granule cells seen in psychotic patents often suggest secretory activity [attributed to granule cells (30)].

Emotional disturbances were common in psychotic patients whose brains we have studied (33). Mood was variable and often unusual. We studied more than a hundred brains which showed a severe mycotic destruction of all layers of nerve cells in the hippocampal gyrus. Severe atrophy of this gyrus occurred. Degeneration of perforating fibers in the subiculum was present. In some of these cases a considerable accumulation of amyloid bodies was seen in the subicular zone which indicated that edema had been present (33). Emotional reactions were noted in the records. Since other cortical areas which connected with the hippocampal gyrus were equally involved by the disease, emotional disturbances indicated a condition more widely spread in the neopallial cortex. Hippocampal pyramids were usually intact, but when they showed disease a euphoric or grandiose state was exhibited by the patients. In some cases of unusual mood, the fascia dentata cells were arranged in the form of reticulum as if their cytoplasm had partly fused around many fluid spaces between them. In deteriorated patients, atrophy of the hippocampal formation was present, and their mood was absent. Epilepsy was not present in most of our cases. In disease of the uncinate cortex near its anterior temporal junction there was some question of the association with reactions of affectionate and erotic behavior. Stimulation of the hippocampus produced violent, emotionally charged reactions in monkeys, and the pick up electrode in the hypothalamus or mamillary body recorded closely spaced high spikes (18, 19, 38, 39).

The fornix, FX, consists of efferent axons from hippocampal pyramids. They enter the alveus on ventricular surface (Fig. 7) pass up under the splenium of corpus callosum where commissural fibers pass to the opposite hippocampus (Fig. 6, FX). Other fibers pass forward over the dorsal surface of the thalamus where some enter the septal nuclei (4, 21). In front of the interventricular foramen, the fornix penetrates the thalamic substance and passes back to the mamillary body in which its fibers end (7, 9). Some fibers are said to pass back in the fornix; they probably reach the fascia dentata as Cajal has illustrated.

It seems obvious that hippocampal discharges are passed on to the hypothalamus and mamillary body (7). That such is the case in emotional disturbances evoked by various predicaments and exigencies of alarm may be assumed (13, 18, 19, 38, 39). The ensuing hypothalamic reactions, as storms out of the hypothalamus, may affect the heart beat, adrenal secretion, arouse the muscular and nervous system to prepare the individual for more than usual exertion. Emotional reactions are accompanied by such bodily changes which are much exaggerated over the normal (Cannon 1930). The motor apparatus is often involved in these episodes. Vagotonic startles, and alimentary disturbances may occur.

Mamillary body, M, is a round mass of small nerve cells which receive the fornix

and give rise to the mamillothalamic tract which ends in the anterior nucleus of the thalamus. Near its origin the fibers of the tract bifurcate and their lower branches form the mamillotegmental tract. This tract passes dorsally in the tegmental region of the midbrain and ends. Stimulation is said to produce a steep rise of blood pressure.

The anterior thalamic nucleus (Figs. 2 and 3, A) receives the main parts of the mamillothalamic tract. Its cells give rise to fibers which pass laterally to enter the internal capsule. They are distributed to the medial, frontal cortex and the cingulate gyrus.

The cingulate gyrus participates in alerting and arousing the individual, in increasing and directing visual perception and emotional activity. Stimulation produces activation of the cingulate and frontal cortex (12, p. 208). This cortex also sends fibers down to the reticular formation (31, 32). Tumors of the corpus callosum shove the midbrain back against the tentorial notch. This interferes with consciousness. It may also crease the uncinate cortex.

The subcallosal gyrus and medial frontal cortex are closely related to the cingulate cortex (Fig. 6). Irritative lesions in this region produce agressive and uncontrolled activities. The tendency to snarl, anger, rage and fight has been noted in experimental animals. Sclerosis of branches of middle cerebral arteries that supply this cortex, olfactory tubercle and posterior orbital cortex are frequently involved in patients with these proclivities. It is probable that other psychopathic dispositions are related to changes in this medial region (42).

The hippocampal system appears to be concerned with emotional experiences and appropriate reactions in other related parts of the brain (7, 13, 16, 18, 29). The emotional aspect of personality may be elaborated here (42).

HYPOTHALAMUS AND HYPOPHYSIS CEREBRI

The hypothalamus may be regarded as the head region for autonomic and visceral regulation, affecting the internal secretions, abdominal and pelvic viscera, cardiovascular system, bodily nutrition and temperature. These functions have been explored experimentally. They support in many ways the needs of inner vegetative life, and the emotional reactions of the hippocampal formation. Hypothalamic nuclei are distinguished as preoptic, supraoptic, tuberal, lateral and mamillary (Figs. 8 and 9) (6, 7, 8, 10, 40).

The preoptic area (Fig. 8, PO) forms a bed nucleus of granule cells for the medial bundle of the forebrain, MF, and terminal stria, TS. As might be surmised the granule cells may facilitate and inhibit cortically induced movements, respiration, blood pressure, etc. The terminal stria from amygdala may be an inhibitory path for calm, docile behavior and possibly sex (12).

Medial bundle of the forebrain arises in the medial cells of the subcallosal gyrus (lower septum). It passes back through the preoptic region of granule cells and lateral hypothalamic nucleus (Figs. 1, 8 and 9, MF). As in lower mammals, so in man, though much reduced, it is an important afferent path to the lateral hypothalamic nucleus, LN. Four other paths of importance are terminal stria, fornix, tegmentohypothalamic and periventricular fibers.

The lateral hypothalamic nucleus (Fig. 8, LN) extends the length of the hypothalamus from the preoptic area to the side of the mamillary body. Here its medial extension is the posterior hypothalamic nucleus, PH. Its main afferent path is the medial bundle of the forebrain which ends among its many multipolar cells. This extensive nucleus may be regarded as the origin of fibers which descend into the tegmentum and terminate on other autonomic, especially parasympathetic cells, in the tegmentum, medulla and spinal cord (20). The autonomic drive reaches the feeding centers (5), vagatonic, vasomotor, cardiac, respiratory, gastrointestinal and pelvic centers. Accelerated activity for defense reactions, feeding, rage, fight and flight may be aroused

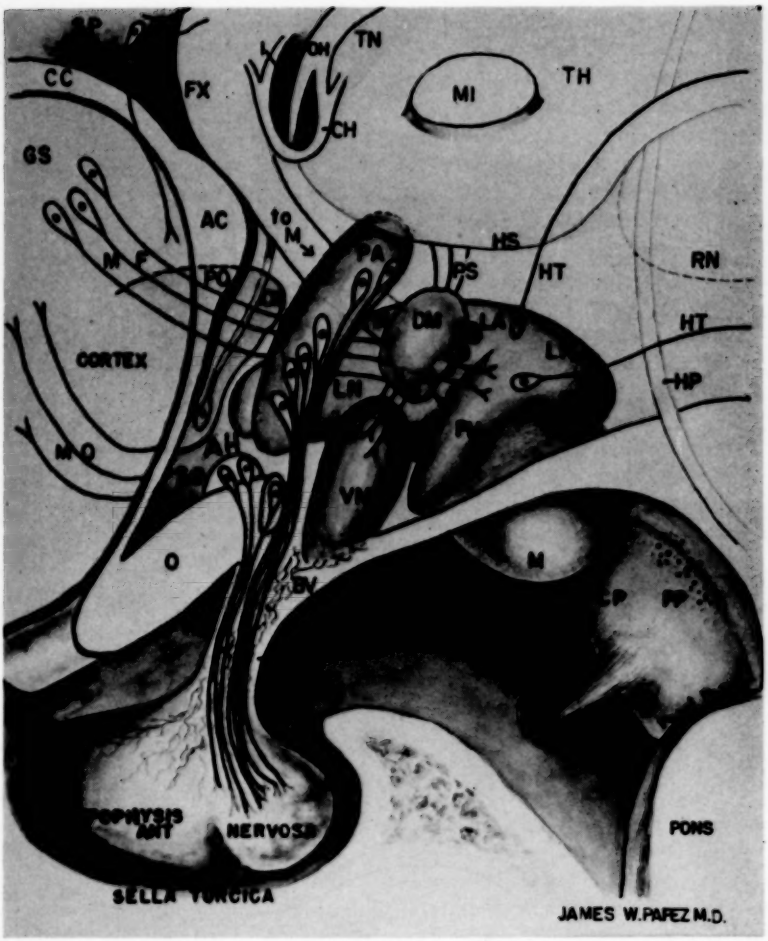

FIG. 8. Hypothalamic nuclei and connections. A drawing of medial view of right hypothalamus showing the position of its nuclear masses and their connections. ×4. LN is large lateral nucleus which receives the medial foregrain bundle, MF, and gives rise to hypothalamic tegmental tracts, HT, to lower autonomic centers. SO is supraoptic nucleus which gets fibers from optic tract, O, and sends fibers to pars nervosa of hypophysis. It is surrounded by a granule cell nucleus AH related to the vascular plexus. BV. PA is paraventricular nucleus connected to the pars nervosa. VM is ventromedial, granule cell, nucleus related to vascular plexus, BV. DM is dorsomedial, granule cell, nucleus. PO is medial preoptic nucleus which borders on PA. BV is the vascular plexus that connects with the anterior hypophysis. It probably carries secretions from AH and VM down to this gland. See Figure 9.

by this system (1). The control for docile behavior may come from the terminal stria and preoptic cells (12). These controls require more study to be sure. Clusters of granule cells, the lateral tuberal nuclei (Fig. 8, LA) occur in the lateral nucleus.

The supraoptic nucleus, SO, consists of two main parts composed of large cells. Their chief afferent connections are a root of fibers from the optic nerve close to which they lie, and terminal stria. Its main efferent fibers pass to the pars nervosa of the hypophysis and terminate among the pituicyte cells and their outer membrane cells applied to the vascular plexus. Granule cells (Fig. 9, AH) of the postoptic area (median emi-

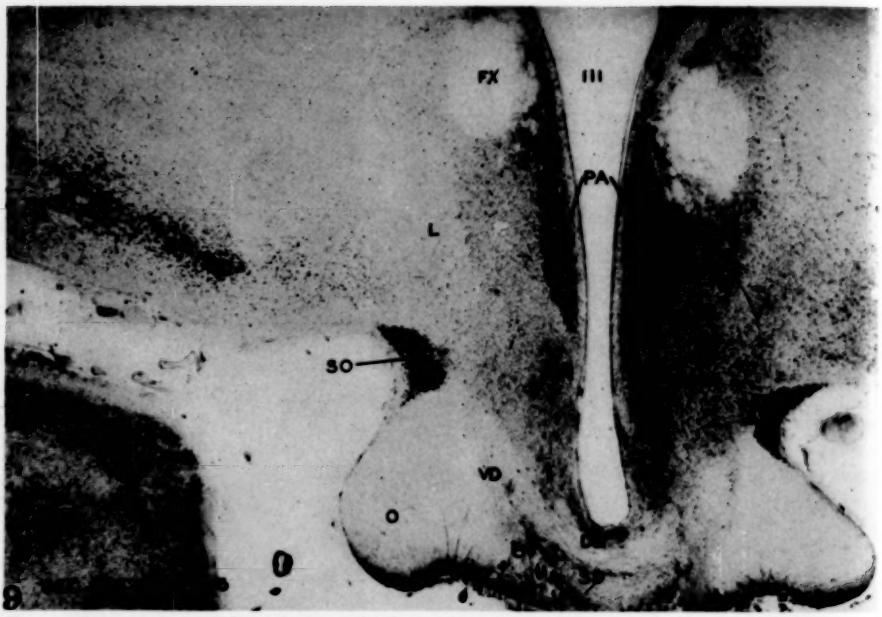

FIG. 9. Supraoptic nuclei. A photograph of a section through human anterior hypothalamic region just posterior to optic chiasma. This shows the relation of granule cells of anterior hypothalamic nucleus, AH, to blood vessels, BV, at bottom of figure. Paraventricular, PA, supraoptic, SO, preoptic, PO, and nucleus basalis, NB, are shown.

nence) surround the nucleus and possibly facilitates its function of water balance, sex maturation, etc. The terminal stria ends among their cells. The close relation of the granule cells AH of the supraoptic region to the vascular plexus, BV, can be seen in Figure 9, AH. One may surmise that these granule cells secrete some cholinergic substance into the nets of the vascular plexus (30).

The paraventricular nucleus has a vertical position close to the ependyma (Figs. 8 and 9, PA). Its large cells send their axons down the hypophysial stalk to the pars nervosa of the gland. A regulation of water balance, electrolytes and carbohydrate metabolism have been suggested as the function of this nucleus. The pars nervosa secretes the antidiuretic hormone which helps to retrieve excess water from the kidney tubules to the blood vessels. The oxytocic hormone is also secreted by this part of the gland. It contracts the gravid uterus to help expel the fetus. The medial preoptic nucleus, PO, is a related mass of granule cells. How these cells function deserves consideration (30).

The ventromedial and dorsomedial hypothalamic nuclei are composed of small granule cells. The ventromedial nucleus is a prominent structure in the human embryos and fetus (28). It receives the pallidohypothalamic fibers, and possibly responds to muscular activity. Stimulation of the anterior part of the temporal cortex close to the visceral area produces large spikes in the ventromedial nucleus (38, 39). This nucleus may produce a cholinergic secretion absorbed by the vascular plexus, BV, which carries the secretion to the pars anterior of the hypophysis. The cells of this gland produce the thyrotropic, adrenotropic, growth, and gonadotropic hormones. When the posterior parts of these nuclei are removed the animal exhibits rage reactions (1, 43). Some

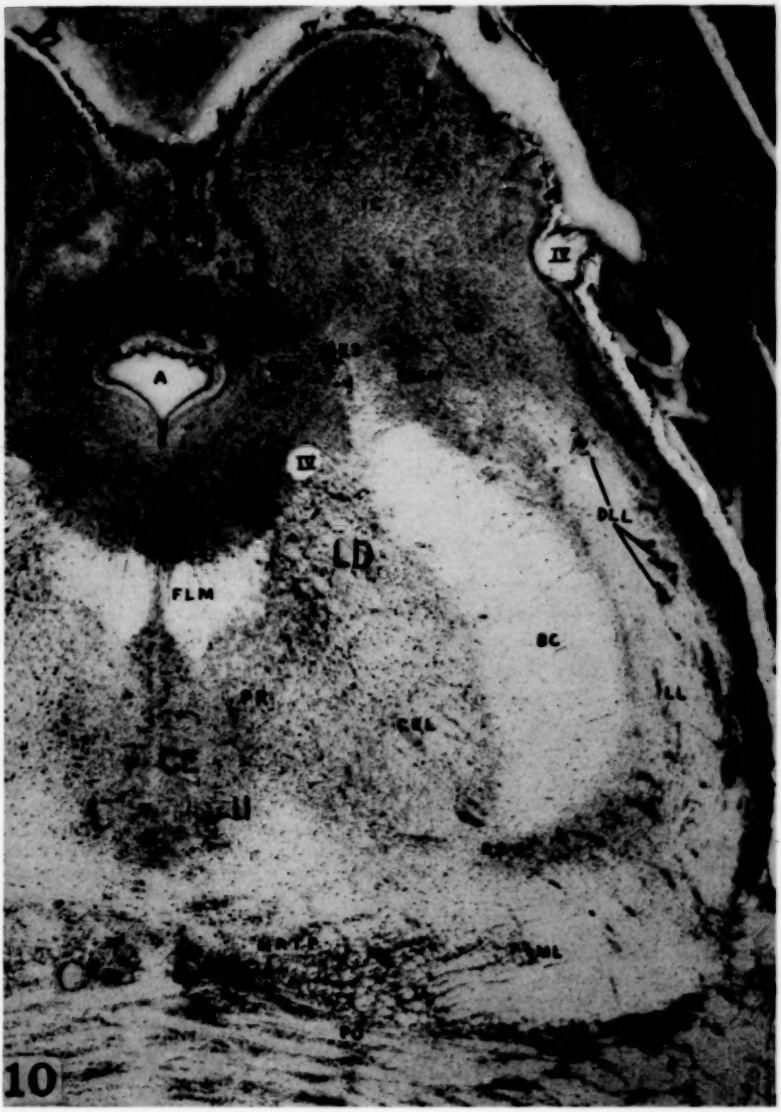

FIG. 10. Centrencephalic nuclei along raphé of central tegmental region of a chimpanzee at level of root of 4th cranial nerve, cresyl echt violet stain. ×4. Central granule cell nuclei, CE, are extensive along midline; nucleus linearis, LI, on each side; on the side lateral, dorsal tegmental nucleus, LD. The midline nuclei are extensive and vary much at different levels. They take an oblique course through the pontine tegmentum down to the level of origin of the medial reticulospinal tracts (25).

form of suppression of rage is implied in these results.

The dorsomedial hypothalamic nucleus DM of granule cells seems to inhibit the feeding reaction which stem from the lateral hypothalamic nucleus. Removal of the medial nuclei releases appetite (5).

The posterior hypothalamic nucleus, PH,

is a medial part of the lateral nucleus. It lies in front of the upper part of the mamillary body. Both of the above nuclei may be influenced by activities of closely related granule cells nuclei. This needs more study. Hypothalamic nuclei are severely damaged in mental deficiency (22), and in psychoses (23).

The hypothalamic nuclei as a whole are concerned with bodily needs, such as demands of nutrition, water balance, internal secretions, growth and sex activities through the hypothysis; body temperature, work and exigencies of economic, social and emotional behavior through the muscular and autonomic nervous systems. The hypothalamus is the caretaker of these basic biologic needs of the individual.

SUMMARY

The olfactory, visceral, hippocampal and hypothalamic neural mechanisms are described. Their interconnections receive special attention. Together they form the visceral brain or limbic lobe. Working together these several parts subserve the basic biologic needs of the individual. Olfaction takes part in primitive search for food, feeding and identification of odorous objects in the environment. A primitive olfactory "motor" pathway from the nucleus basalis to the habenula, thence to interpeduncular nucleus, to tegmental and reticular formation is illustrated. The visceral cortex, claustrum, uncinate bundle and amygdala are described with some interpretation. Their relations to the original olfactory mechanisms, internal capsule, and basal ganglia are indicated. The hippocampal formation, as a cortical dependency, and its mechanism for integration of emotional states, arousal to action, hypothalamic and cortical responses are considered. Hippocampal, neuronal mechanism is illustrated. The hypothalamic nuclei, their amygdalar, forebrain, hippocampal, autonomic and hypophysial connections are illustrated and defined. It is pointed out that large-celled nuclei conduct nerve impulses; whereas the granule cell nuclei may possibly have a cholinergic function. Most of the reactions of the visceral brain are biologically grounded, close to the reflex-like level of innate activities. They are designed to preserve, maintain and protect the individual, and control his vegetative needs, an important segment of inner life. From this point of view each mechanism appears to have an appropriate structural design.

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Explanation of Letters on Figures

A = Anterior nucleus of thalamus
AB = Accessory basal nucleus of amygdala
AC = Anterior commissure
AH = Anterior hypothalmic nucleus
AM = Amygdala
AN = Angular bundle
AP = Anterior perforated substance
AS = Association bundle

AT = Amygdalar tract
B = Basal nucleus of amygdala
BN = Bed nucleus of terminal stria
BV = Blood vessels
C = Central or bed nucleus of AT
CA = Caudate nucleus
CC = Corpus callosum
CE = Central superior nucleus
CH = Corticohabenular tract
CO = Cortical nucleus of amygdala
CL = Claustrum, layer vi
CP = Cerebral peduncle
CR = Internal capsule, corona radiata
CS = Central sulcus
D = Diagonal band
DD = Dorsal supraoptic decussation
DM = Dorsomedial nucleus of hypothalamus
DR = Dorsal nucleus of thalamus
DT = Dorsal tegmental nucleus
EX = External capsule
FD = Fascia dentata
FX = Fornix
G = Granule cells
GC = Gyrus cinguli
GO = Orbital gyrus
GS = Gyrus subcallosus
H = Hippocampus
HA = Habenular nuclei
HG = Hippocampal gyrus
HP = Habenulointerpeduncular tract
HS = Hypothalamic sulcus.
HT = Hypothalamicotegmental tract
HY = Hypothalamic nuclei
I = Interventricular foramen
IC = Inferior colliculus
IF = Infundibulum
IN = Interpeduncular nucleus
i, ii, iii, iv, v, vi = Layers of cortex
INS = Insular cortex
IP = Inferior thalamic peduncle
L = Lateral nucleus of amygdala
LA = Lateral tuberal nuclei
LN = Lateral hypothalamic nucleus
LO = Lateral olfactory nucleus
LD = Lateral dorsal tegmental nucleus
LS = Medial longitudinal stria
LT = Lateral olfactory tract
M = Mamillary body
MF = Medial forebrain bundle
MI = Massa intermedia
MO = Medial olfactory tract and area
MT = Mamillothalamic tract
NB = Nucleus basalis in AP
NH = Nerves to hypophysis, pars nervosa
O = Optic tract, or chiasma
OB = Olfactory bulb
OH = Olfactohabenular tract
OR = Orbital cortex, or radiation
OT = Olfactory trigone, or tubercle
P = Pineal body
P_1 = Inner division of pallidum
P_2 = Outer division of pallidum
PA = Paraventricular nucleus
PC = Posterior commissure
PF = Perforating fibers
PH = Posterior hypothalamic nucleus
PO = Preoptic area, part of AP
PP = Posterior perforated substance
PS = Pallidohypothalamic fibers
PT = Ptamen
R = Reticular nucleus of thalamus
RN = Red nucleus
SB = Subiculum, mostly PF
SC = Superior colliculus
SO = Supraoptic nucleus
SP = Spetum pellucidum
SU = Subthalamic nucleus, or decussation
T = Tegmentum
TH = Thalamus dorsalis
TN = Parataental nucleus
TP = Temporal cortex, area 38
TS = Terminal stria
TV = Temporal ventricle
U = Uncus of hippocampus
UC = Uncinate cortex
UF = Uncinate fasciculus
VM = Ventromedial nucleus of hypothalamus
3,5 = Cranial nerves

EFFECTS OF SEPTAL AND AMYGDALOID LESIONS ON EMOTIONAL BEHAVIOR AND CONDITIONED AVOIDANCE RESPONSES IN THE RAT[1]

FREDERICK A. KING, PH.D.[2]

Experimental destruction of the septal region of the forebrain grossly alters responses to stimuli that normally do not provoke affective excitement in the rat. Postoperative behavior consists of extreme muscular tenseness and of violent attack or flight reactions of a degree, rarely, if ever, seen in the normal animal. Brady and Nauta (3) were the first to observe such behavioral changes, and their observations suggest that normally the septal area exerts an inhibiting influence on display of fearful and aggressive emotion.

Affective behavior associated with damage to the amygdaloid nuclei have been investigated in cat and monkey, but the results have not been entirely consistent. Bard and Mountcastle (2) found a dramatic increase in aggressive behavior following lesions of this complex in cats, but Schreiner and Kling (8) observed placidity with similar lesions in the same species. Kluever and Bucy (6) report that lesions invading the amygdaloid complex in monkeys also produce placidity, but more recently Rosvold, Mirsky, Allen and Pribram (7) report that following amygdaloidectomy monkeys may show either an increment or decrement in their display of aggressive behavior depending upon whether they are placed in an isolated or group situation.

From the experimental evidence it is clear that the septal region and amygdala both play a vital, if not thoroughly understood, role in affective expression and deserve further study. It was the purpose of the present investigation to supplement previous studies of learned behavior in septal preparations (3, 4) and to extend the systematic investigation of affective changes in amygdaloid preparations to the rat, evaluating the effects of such brain injury upon fear- or anxiety-motivated responses. To this end, measures of rate of acquisition and latency of a conditioned avoidance response were obtained from rats with septal and amygdaloid damage. The technique of ratings of emotional responses by observers was employed in the study of overt emotionality in these animals.

METHOD

SUBJECTS

Twenty-five adult male rats of the Lashley strain were divided into four groups in the following manner: (a) 10 unoperated controls, (b) 5 septal operates, (c) 5 amygdaloid operates, and (d) 5 cortical operates (operated controls).

AVOIDANCE CONDITIONING

Conditioning was carried out in a double-grill box composed of two compartments separated by a partition containing an opening through which the animal could pass.

[1] Read before the Annual Meeting of the Society of Biological Psychiatry, Atlantic City, June, 1957. This paper is based in part upon a dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy at the Johns Hopkins University, and upon further work completed at Ohio State University. The author wishes to express his gratitude to Professors Clifford T. Morgan and James Deese of the Johns Hopkins University, and Dr. Joseph V. Brady of the Army Institute of Research, Walter Reed Medical Center, for their valuable assistance and suggestions. Special appreciation is due Dr. Walle J. H. Nauta of the Army Institute of Research, Walter Reed Medical Center, for his generous help in the examination of brain tissue. The assistance of Mr. Herman Teitelbaum in making ratings of emotional behavior is gratefully acknowledged.

[2] The author was a National Institute of Mental Health Research Fellow of the United States Public Health Service during this investigation; now Assistant Professor of Psychiatry, Research Division, Columbus Psychiatric Institute and Hosptial, Ohio State University College of Medicine, Columbus, Ohio.

A buzzer was attached to the ceiling of the apparatus between the two compartments. The floor of each compartment was a brass grill through which an electric shock could be delivered to the feet of the animal. The value of the electric current was measured for all animals and fell within the range of 0.10 ma. to 0.25 ma.

Each conditioning trial began with the sounding of the buzzer. The buzzer was presented until the animal crossed into the opposite compartment. If, at the end of 10 seconds, the animal had not responded correctly, shock was applied to its feet and continued, along with the buzzer, until crossing occurred. The criterion for conditioning was 9 avoidance responses out of any 10 consecutive trials. Inter-trial periods were randomly varied from 60 to 180 seconds.

Conditioning trials were carried out on two consecutive days. On the first day, each animal was permitted a maximum of 30 trials. Those animals not reaching criterion within this limit were returned to their living cages; conditioning was resumed the next day and continued until criterion was reached. For those animals that reached criterion during the first day, conditioning was stopped upon completion of the last criterion trial and resumed the following day until criterion was again reached.

RATINGS OF EMOTIONALITY

A six-component rating scale was used in evaluating the behavior of every animal. The scale was adapted, with certain modifications, from one constructed by Brady and Nauta (3). The six components of the rating procedure were as follows: (a) attack or flight reaction to a pencil presented visually to the S's face, (b) startle and flight reaction to the rat's face, (b) startle and flight reaction to a light tapping on the animal's back with a pencil, (c) resistance to capture, (d) muscular tension and resistance to handling, (e) vocalization reaction during testing, (f) urination and defecation during testing. Each of the above components was evaluated on a six-point scale, and an overall daily score was assigned each animal by adding up the scores obtained for each component of behavior. Two raters independently scored each animal in the living cage. A reliability coefficient between the daily scores of the two raters was computed at +0.92.

All subjects were rated for a total of 9 days. Those animals undergoing surgery received 3 days of preoperative ratings and 6 days of postoperative ratings.

SURGERY

All surgery was performed under the general anesthetic, pentobarbital sodium, injected intraperitoneally. Following incision of the dorsal surface of the scalp, trephine holes were drilled bilaterally in the skull directly over the points where the lesions were to be made. Bilateral electrolytic lesions of the septal region, amygdaloid nuclei, and neo- and cingulate-cortex (operated controls), were made by means of a stereotaxic instrument previously described (9). A platinum-iridium electrode, insulated except for ¾ mm. at the tip was lowered into the brain and a direct current of 2 ma. passed for 30 seconds. A rectal cathode closed the circuit. Upon removal of the electrodes, the incision was sutured with surgical silk. Penicillin was administered intramuscularly.

HISTOLOGY

The brain of each operated animal was fixed in formalin, embedded in paraffin, and sectioned at 20 micra. Every fifth section was mounted, stained with thionin, and studied to determine the locus and extent of the lesion.

RESULTS

AVOIDANCE CONDITIONING

An analysis of the data presented in Figure 1 indicates that septal preparations learn the avoidance response to criterion

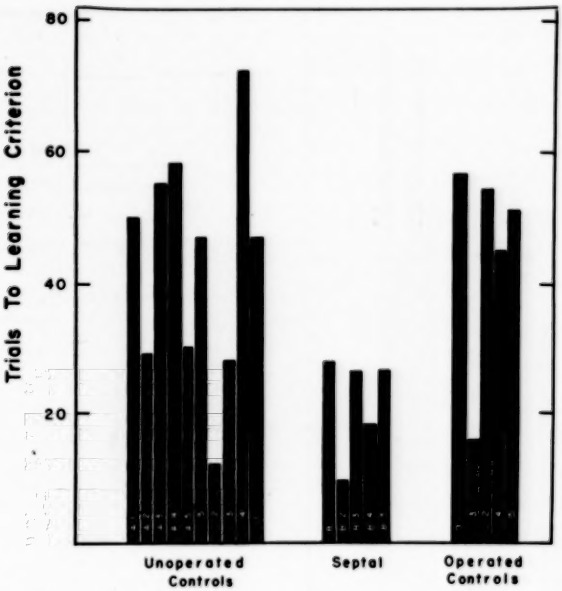

FIG. 1. Rate of avoidance conditioning in unoperated controls, septals, and operated controls.

more rapidly than do either unoperated or operated controls. The difference in conditioning rates between the septals and both control groups was significant beyond the 0.05 level of confidence ($t = 2.48$). Amygdaloid rats did not differ from unoperated or operated controls in rate of acquisition of the avoidance response.

An analysis of the mean latencies on conditioned responses showed no differences between septal preparations and their controls. It is evident from Figure 2, however, that amygdaloid animals show longer response latencies during acquisition of the avoidance response than do their operated or unoperated controls. This difference is significant at the 0.001 level of confidence ($t = 12.81$). It appears that although amygdaloid animals learn the avoidance response as rapidly as do normals in terms of trials to reach criterion, they do not respond as rapidly on individual trials to the conditioning signal.

EMOTIONALITY RATINGS

Figure 3 shows the mean daily scores on the rating scale for emotionality for each of the four groups of subjects: septals, amygdaloids, operated controls, unoperated controls. Preoperatively there are no significant differences among the groups, but it is clear that upon postoperative testing the septals exhibited increased emotionality. A comparison of mean pre- and postoperative scores showed that for septal animals, postoperative scores were raised significantly over preoperative scores at the 0.001 level of confidence ($t = 9.36$), while the scores of the unoperated controls and operated controls were not altered significantly from pre- to postoperative ratings. In addition, on postoperative days the septals differed significantly from the unoperated and operated control groups at a confidence level of 0.001 ($t = 12.67$).

It is evident that emotional expression, as measured by the rating scale employed here, increases dramatically following damage to the septal region. Although amygdaloid operates tend to be lower in emotionality than the control groups, the difference does not reach statistical significance.

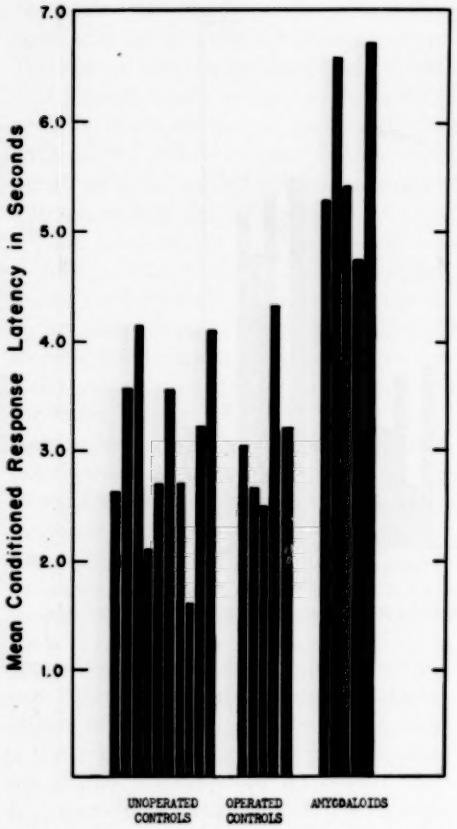

FIG. 2. Mean latency on conditioned responses for unoperated controls, operated controls, and amygdaloid preparations.

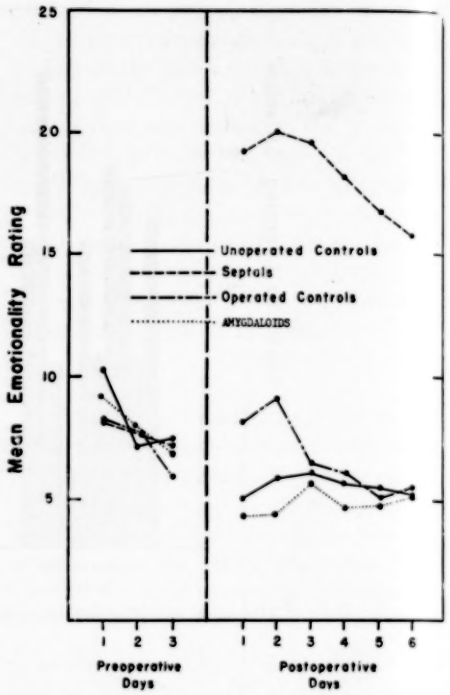

FIG. 3. Mean daily ratings of emotionality for septals, amygdaloids, operated controls, and unoperated controls.

LESIONS

The brains of those animals designated "septals" were found upon microscopic examination to have sustained extensive damage to the septal region. Massive destruction of the precommissural portion of the septal area was evident in all cases, indicating severe interruption of the precommissural fornix system. Heavy damage of the supracommissural septum was present in each case. In many instances, however, the post commissural fornix columns had been left intact, and the anterior commissure was undamaged in all cases. Figure 4 is a photomicrograph of a brain section with typical septal damage.

All animals designated "operated controls" suffered damage of the cingulate and adjacent neocortex. A photomicrograph of a section of the brain of a control operate is shown is Figure 5.

All subjects designated "amygdaloids" suffered bilateral damage to one or more of the nuclei of the amygdaloid complex. Most frequently this damage was restricted to the lateral and basal nuclei, although occasionally partial destruction of central, medial, or cortical nuclei was present. Figure 6 is a photomicrograph of damage to amygdaloid nuclei.

DISCUSSION

It seems clear that extensive damage to the septal region increases the rate of learning an avoidance response and heightens overt emotionality. The increased emotionality agrees with the findings of Brady and

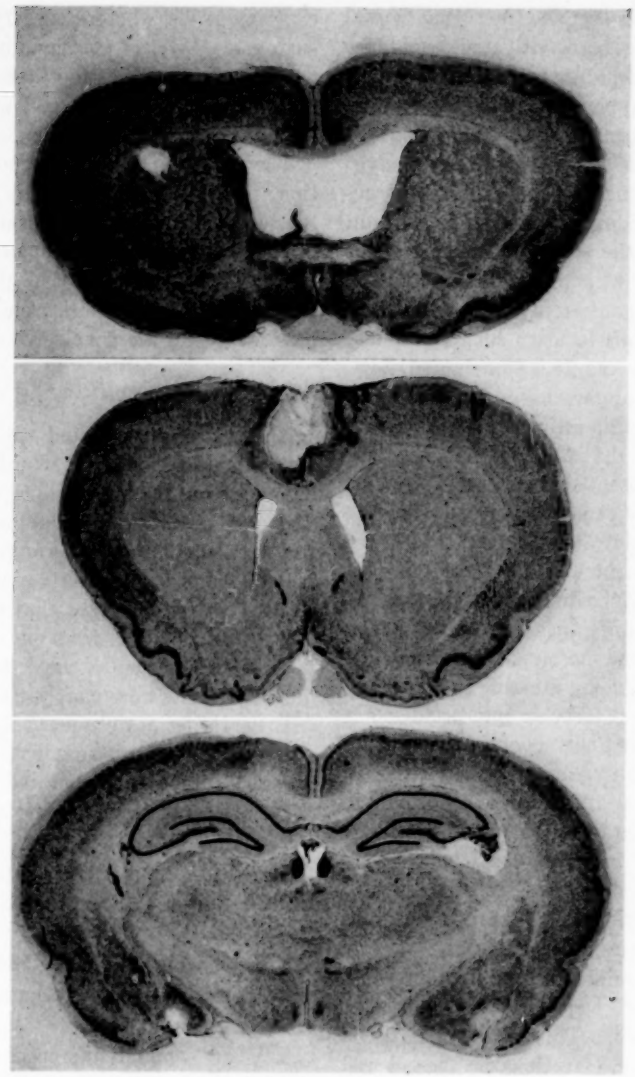

FIG. 4 (top). Brain section of rat with lesion in the septal area.
FIG. 5 (center). Brain section of operated control rat with lesion of cingulate cortex and adjacent neocortex.
FIG. 6 (bottom). Brain section of rat with lesion in the amygdaloid complex.

Nauta (3). These investigators, on the other hand, did not obtain any difference in rate of learning between their septal and control animals. Their learning task, however, was considerably simpler than the avoidance learning studied in the present task. The conditioned emotional response in their experiment consisted of "freezing" and other signs of emotionality in response to a clicker which had been paired in training with shock. It is suggested that this conditioned response is too elementary to differentiate septal and control animals, as it was possible to do in the present experiment with an

avoidance response. The fact that septal animals acquire fear-motivated avoidance response more rapidly than normal animals fits in with the hyperemotionality and panic-like behavior seen in these animals.

No differences appeared between amygdaloid and normal rats in the rate of acquiring an avoidance response. This result contrasts with the study of Brady and his associates (5), who report that cats subjected to amygdalectomy require significantly more trials to learn such a response than do normal cats. It is to be noted, however, that the apparatus and method employed by these investigators seems a considerably more difficult one than the task used in the present experiment. The mean number of trials required normally for cats to reach criterion was over 100; that in the present experiment was only 35. It is possible, again, that the difficulty of the task accounts for the difference in results. In this case, their method more readily permits differences in rate of conditioning to appear.

Some evidence for this interpretation is to be found in an analysis of the latencies of response for amygdaloid rats. Although such rats were no different from normals in trials required to learn, they were considerably slower in responding to the warning buzzer. Such a difference in latencies may indicate an inferiority in learning that might have been more pronounced had the learning task been more difficult. On the other hand, it might indicate a general sluggishness or decline of activity. This has been reported in amygdaloid preparations by Anand and Brobeck (1), though it was not observed by the present experimenter.

Amygdaloid lesions did not alter overt emotionality to a statistically significant degree. These results are not in line with those reported in other species, (5, 6, 8), and it may be that the rats used in the present experiment were before surgery already at such a low level of emotionality that post-operatively there was too little distance remaining at the bottom end of the rating scale to permit differentiation.

The results of this study support the notion, previously expressed by others, that the septal region normally exerts a calming or "dampening" influence upon affective behavior. The hypothesis that the amygdala plays a counterbalancing role in such behavior receives less conclusive support.

SUMMARY

A study was conducted with rats to investigate the effects of septal and amygdaloid lesions upon avoidance conditioning, and overt emotional behavior. The latter was measured on a six-component rating scale. Conditioning was carried out in a shuttle box, so arranged that the rat could move through a door from one compartment to another to avoid a shock signaled by a buzzer. Results were as follows:

1. Septal lesions produced a dramatic increase in overt emotionality, whereas unoperated controls and operated controls with neocortical and cingulate lesions showed no such damage.

2. Septal lesions decreased the number of trials required to learn the avoidance response.

3. Lesions in the amygdaloid nuclei of normal animals did not significantly alter overt emotionality.

4. There were no differences in rate of acquisition of conditioned responses between normal rats and rats with amygdaloid damage. However, the response latencies of the amygdaloid preparations were significantly lengthened. Throughout the experiment, no other group differed from normals in response latency.

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THE SEVERELY DEFECTIVE INDIVIDUAL[1]

SEYMOUR B. SARASON, Ph.D.[2] AND THOMAS GLADWIN, Ph.D.[3]

A. PSYCHOSIS AND MENTAL DEFICIENCY

The relationship between psychosis and subnormal functioning is a research problem about which a fair amount has been written over the years. At different times comprehensive reviews of the literature and critical discussions of the problem have appeared (4, 7, 8, 23, 24, 26, 28, 31, 32, 45–49, 58, 59) which with almost no exception have made a plea for a systematic study of the problem. Unfortunately, as with so many other research problems in subnormal functioning, this theoretically and socially important problem has not attracted the attention of more than a few researchers. It would seem appropriate, therefore, to summarize briefly some of the things that seem to be known about the relationships between psychosis and subnormal functioning.

1. Psychosis is found at all levels of subnormal functioning. Even in the case of idiots marked and dramatic psychotic-like changes in behavior have been noted; in some cases, apparently, the behavior is episodic while in others it is followed by deterioration.

2. Psychosis occurs in cases with no discernable organic pathology as well as in those with marked pathology.

3. Practically every major psychotic symptom which has been described in the non-defective patient has also been noted in many of the defective cases. There appears to be little justification for the generalization that when psychosis occurs in a mentally defective individual it is necessarily less "complex" than when found in the non-defective individual. There is also little support for the equally sweeping generalization that psychosis or psychotic-like behavior in the defective individual tends to be of short duration.

4. As a result of work in the last twenty years on childhood schizophrenia, there is little doubt that many children who were committed to an institution for the mentally defective were in fact misdiagnosed and misplaced. Kanner's (33–38, 53) now classic descriptions of the autistic child provided further evidence that our institutions contained cases in which personality or affective rather than intellectual maldevelopment was the primary factor. While one would like to believe that our institutions are admitting far fewer of these cases, there is no evidence that this is the case.

5. The schizophrenic type of reaction is the most frequently found psychosis among the mentally defective.[4]

[1] This article contains two of ten chapters of a report ("Psychological and Cultural Problems in Subnormal Functioning") prepared for the National Association for Retarded Children. The full report will be published in the February, 1958, issue of the Genetic Psychology Monographs. In the two chapters we have quoted rather extensively from clinical sources because the kinds of problems herein discussed are not clinically familiar to many workers.

A companion report on biological factors has been prepared by Dr. Richard Masland and will be published in the American Journal of Diseases of Children.

[2] Yale University, New Haven, Connecticut.

[3] Community Services Branch, United States Public Health Service.

[4] In Pollock's (47, 48) study of mental disorders among mentally defective first admissions to the state hospitals of New York, approximately 40 per cent of the cases were of the episodic variety. The cases in this study probably are made up of individuals who resided in the community and those who were transferred from institutions for the mentally defective. It seems reasonable to assume that the chronically psychotic, mentally defective individual who has made some kind of adjustment in the community or institution would not be sent to a state hospital. The episodic variety—which may be characterized by excitement with depression, paranoid trends or hallucinatory attacks—usually present such supervisory problems to the community or institution that they are likely to be sent to the state hospital. If a more representative sample of the mentally defective population were studied, it would not be surprising if the proportion of chronic cases would be more than 20–25 per cent reported by Pollock.

6. The incidence of mental illness among the mentally deficient appears to be much higher than in the general population.

The theoretic and research implications of the above summary statements—which undoubtedly are not of equal status in terms of scientific validity—cannot, in our opinion, be discussed without serious consideration of the problem to which we now turn.

The Problems of Diagnosis and Incidence

When we are confronted with the possibility of the diagnosis of psychosis in a non-defective individual, several questions are routinely asked:

How does the individual view and experience his environment and how deviant is this from the way in which we (or others in his particular cultural group) describe that environment? What is his conception of himself (*e.g.*, ability, powers, attractiveness, usefulness, worthfulness) and how is this related to his conceptions of others in his environment? In asking these questions one is interested not only in the contents of an individual's views and experiences but in the kinds of thought processes which they indicate: the bases of his conceptualization, the nature of his associative processes, the degree and nature of fantasy, the role of language in communication, etc. Put most briefly, these questions are directed to answer the question of the nature and adequacy of the individual's interpretation of reality.

What is the nature and role of emotions and feelings in the individual's adjustment and thinking? How are these factors related to the questions raised above? What may be inferred about the role and adequacy of the defensive reactions associated with strong feeling and emotion?

What light is shed on the above questions from an examination or reconstruction of the life-history? What are the experiences (*e.g.*, separation, trauma, failure) or types of relationships (*e.g.*, social class, ethnic) which influences the development of the individual and are reflected in his current life adjustment?

The above are certainly not all the questions that are asked when one is faced with the diagnostic problem of psychosis or, for that matter, understanding the personality of *any* individual. But we think we have listed enough of the questions to substantiate the conclusion that the diagnostic process is a searching, comprehensive investigation which has the understandably ambitious aim of attaining understanding of another person's "psychology"—*that* individual's way of thinking, feeling, and acting. *Rarely, if ever, is the mentally defective individual studied or viewed in this way.* We think it would indeed be an interesting research project to obtain and evaluate diagnostic folders in the files of institutions for the mentally ill and the mentally defective. The important point for our present discussion is that since the mentally defective individual is rarely studied in the way indicated above, we cannot at all be secure in accepting available statistics on the incidence of psychosis in the mentally defective population. The absence of adequate and appropriate diagnostic procedures suggests that available statistics are probably largely based on the obviously psychotic individual and that the less flagrant case goes unnoticed or his "peculiar" behavior is blamed on his I.Q. (When an individual with an I.Q. of 180 behaves peculiarly we do not assign cause to his I.Q., but should an individual with an I.Q. of 50 behave similarly we usually point to the I.Q. as the etiologic agent.)

It should not be thought that if one could carry out adequate diagnostic procedures with a representative sample of the mentally defective population, the true incidence of psychosis would be easily established. One of the most thorny diagnostic problems involves the lower grades (*i.e.*, idiot and imbecile) of mental deficiency. In many of these cases language is either non-existent or inadequate for communication, the establishment of rapport difficult, and a wide variety of organic pathologies present. In short, the conditions which ordinarily allow us to understand the covert reactions and

experiences of another person are present minimally with many of these cases. It is usually when marked and dramatic changes in the overt behavior of such individuals are observed, that we can deduce that some kind of corresponding change has taken place in the covert aspects of behavior. The following cases from Greene (23) are examples of this:

In regard to the symptomatology of psychoses occurring in even the Low Grade Mental Defectives, it is a very common expression heard among the laity and I frequently hear it from the medical profession, including psychiatrists, that such and such an individual is an "insane idiot." Just what is meant by this term, I do not comprehend fully or fully understand what they comprehend by making such a diagnosis and think it a rather loose terminology, for certainly all idiots are not insane, neither all insane, idiots. It is my belief, however, that we do have actual psychosis appearing in the very low-grade mental defective and they do deteriorate. The following is an example of psychosis in an individual who might be definitely classified from the standpoint of mental defect as an idiot.

S. M. Chronological age, 37 years. Mental age, 2.6. I.Q. 16. This individual is the youngest of 7 children, two of whom have been patients at this institution, both feebleminded. Other than sibling's history of mental defect, there is no other family history of note. Physically, she is underheight but generally well-nourished, with rough coarse skin, dissimilar ears of simple pattern and with adherent lobules. She has a high-narrow palate, facial asymmetry rather marked, otherwise no marked stigmata. Developmental history indicates that she began to walk at the age of one year and to talk at three years, but uses only phrases at the present time. According to developmental history, in childhood, was obedient, apparently good-tempered and able to learn to do simple errands. Admitted to this school at the age of fourteen, she immediately began to show marked emotional instability in laughing one moment and crying the next, apparently without external stimulus; always exhibited a tendency to collect bits of rubbish and articles of clothing which she secreted about her person and this habit has continued to increase. Had long periods of crying at the age of 27 without apparent causative factors and at this age had to be excluded from the physical training classes because of her noisy and erratic behavior. She shows increasingly frequent periods of restlessness, sleeplessness, wandering and running about the house in an aimless manner between periods of sitting motionless under a piano or lying under a bed. During these excited periods she refuses to go to the dining room for meals and will tear her clothes in order to avoid it. She continues to cry without reason almost continually and must be coaxed and wheedled to do those things which she formerly performed without question. She has lost the ability to do tasks which she used to accomplish well, such as bed-making.

These episodes of excitement are usually accompanied by loss of weight. Between attacks, she seems to be in good physical condition, eats well and is generally quiet. The attacks of excitement are becoming more frequent and lasting for longer periods. In this case, the individual presents the differential diagnosis from the usual idiot in that she has episodic psychotic attacks, loss of weight, emotional instability, excitement and depressions, refusal of food, collecting of purposeless and useless rubbish, sleeplessness, and furthermore, she has deteriorated.

The following is an example of a low-grade imbecile with psychosis.

J. B. Chronological age, 42 years. Mental age, 4.6. I.Q. 28. This boy is a sturdy imbecile with the cyanotic hands, dissimilar ears, and facial asymmetry so often associated with mental defect. In addition, it is recorded that his eyes are dissimilar in size and shape; his speech is clear, and there is a peculiar twitching of head and extremities. He is somewhat under average height, with a very good muscular development.

The family background is very poor, the mother mentally deficient and a patient in the department for the insane at the Tewsbury State Infirmary, and the father a chronic alcoholic. The patient is the last of 6 pregnancies, 5 having died, 4 of hydrocephalus and 1 of phthisis.

There is very little definite information concerning early development, but the history states that the peculiarity was evident from birth. He never learned to dress or undress completely, and though indolent, has always been obedient

and amenable to discipline. When admitted to the institution he knew neither colors nor numerical combinations, could not be trusted to do the simplest errand or any sort of useful work.

Since his admission, most of the time has been spent at the farm colony, where he has learned some of the rudimentary tasks in connection with farming, but his work is not entirely satisfactory, for he is clumsy, erratic and undependable. His customary good nature has been increasingly interrupted by periods of violent excitement. During these episodes he exhibits the stereotyped attitudes and jerky, monotonous speech characteristic of dementia praecox. In 1926 these traits became more bizarre in character; he began to talk to himself and developed a rather elaborate ritual in regard to eating. The matron reports that at times he would remove his clothing and rush out of doors, irrespective of the weather; again, would pour hot liquid food over his head, and several times deliberately leaned against hot steam pipes.

There is a regular periodicity about these attacks, the patient being quiet and well behaved for two weeks and disturbed and violent for the succeeding two.

These episodes became so increasingly severe that it was necessary to remove him from our farm colony and care for him in the infirmary of the institution proper and at the present time, he has changed from a pleasant, agreeable, rather talkative imbecile to one who wanders around by himself, rolls his head from side to side, has an anxious, apprehensive expression, talks to himself, has had an entire change of disposition. He exhibits erratic behavior, and instead of being able to help in the usual routine, requires constant supervision in order that he may be even clothed. He is very untidy in his habits, profane and obscene in his language, queer in his mannerisms about eating, each spoonful of food that he takes from his plate has to be passed under his knee and into the other hand before being put in his mouth. He is now a muttering, suspicious, erratic and depressed individual, with deterioration, and was committed April 23rd to a mental hospital.

Perhaps the most extensive clinical investigation of psychosis in the lowest grades of mental defect was that by Earl (15) who described what he called the "primitive catatonic psychosis of idiocy." From a study of 135 male idiots he differentiated a group of 38 cases whom he thought fitted in this category. The following behavioral descriptions are from Earl:

SIGNS OF DETERIORATION

A general deterioration of behaviour and of adaptation is constantly present. Speech fades out, toilet habits are lost, and the patient becomes apathetic, inaccessible, degraded, and destructive. Reliques of former abilities can often be demonstrated in grossly deteriorated patients. Thus several cases in this series show an understanding of speech quite out of keeping with their apparent mental level. Others can dress or undress or perform simple tasks: two of the cases can play simple ball games well, though they are quite untrainable and require every nursing care and attention. Even on the purely neurological level these people often show a degree of co-ordination and control complexity of movement quite beyond the pure profound idiot.

Many of the symptoms of deterioration of behaviour can be analysed into catatonic manifestations or signs of emotional dissociation, and will be discussed under these headings. It seems probable, however, that a certain degree of permanent loss of potential ability is always present.

SIGNS OF CATATONIA

Catatonia is a marked feature. The cases may be roughly divided into two groups—the stuporose or cataleptic, and the excited or hyperkinetic. There is no sharp line of demarcation between the groups; it would be more accurate to speak of a series, with the completely apathetic and the completely excited cases at either end. Twenty cases were mainly cataleptic and eighteen mainly excited in type.

Catalepsy. That loss of motor initiative described by De Jong and Baruk in their experimental animals is well seen in the cataleptic group. These patients stand about in the typical posture of the schizophrenic stupor, with bent head and semiflexed knees—hands hanging idly by the sides. The patients take little or no notice of threatened danger, but they will move when ordered to do so and, having once started, they will continue at a familiar simple movement. Thus they may be herded round a ward, or even engaged in very simple tasks such as cleaning spoons.

Automatic obedience. Echolalia is not uncommon but is rarely marked. Occasional echopraxia is also seen, and the understanding for words can sometimes be demonstrated in a patient who is allegedly "incapable of speech or of understanding speech"—for a sharp command, unaccompanied by gesture of facial expression, may be at once obeyed.

Muscular catatonia. The actual muscular catatonia varies. It could be demonstrated in twenty-three patients. It is commonest in the simple cataleptic group. In the hyperkinetics negativism is the rule, and muscular rigidity difficult to demonstrate.

The irregular type of stretching reactions described by Claude, Baruk, and Nouel was very well demonstrable in many cases. During passive extension of the flexed arm, the biceps tendon can be felt to resist, to contract, to move easily, even to anticipate movement; each for a moment in turn and quite irregularly. In some of the cases the anticipation of passive movement was obvious and the flexed arm extended itself in advance of the movement of the examiner. Lastly, in five cases after passive flexion and extension of the forearm, rhythmic repetition of the movements was set up which lasted for several minutes.

The degree of muscular catatonia present does not correlate exactly with the degree of psychosis present. This was well seen in one case in which the rhythmic repetition was continued for some minutes during which time it was possible to persuade the patient to attend to and even to reply to questions.

Autism is seen in all the cases, and is sometimes marked. The patients are not interested in their surroundings; are often solitary, sometimes morose, refuse to mix with others and tend to conceal themselves under tables or in corners. The head is often bowed, or the coat pulled over the face; or the boy may sit in the "intrauterine" position.

Mutism. Some degree of mutism is always found. It varies from one patient to another and simetimes from day to day in the individual patient. Some patients will answer questions; some have a few stereotyped words and phrases; some remain quite mute. This mutism seems to be largely an autistic or negativistic feature rather than a true loss of ability. And here we have evidence that, in some cases at any rate, the mutism cloaks the degree of knowledge of, and contact with, reality. Thus one boy of twelve years who had been grossly psychotic since his second year, who had been partially mute for years, and who had spoken no word during the three months he had been under observation, managed to exasperate an attendant into threatening to smack him; to which he replied with great emphasis "No! you bloody well won't!" ...

SIGNS OF EMOTIONAL DISSOCIATION

Variability. The emotional abnormality shows itself in various forms, some of which have already been described under other headings. A rather wide variation in the general reaction to life frequently occurs; a matter of months or even years at a time. More obvious is the occurrence of so-called "episodes" or "mental turns" lasting for a day or a week. These episodes may take the form of excited periods or states of depression, weeping or apathy, or vague anxiety. One of the hyperkinetic cases will weep steadily for two days at a stretch; another has days on which he is almost somnolent; whilst some of the mildest of the cataleptic group have their days of excitement and activity.

These episodes may be very early signs and may occur as the only emotional abnormality seen in otherwise fairly "normal" idiots. They are possibly analogues of the psychotic episodes which occur in some high-grade aments, and which, when frequently recurring, sometimes presage the permanent installation of a psychosis.

Impulsiveness. Sudden outbursts of general excitement sometimes occur, lasting a few minutes or more, often accompanied by hyperkinesis and occasionally by manifestations of fear, rage or pleasure.

These outbursts are closely related to the hyperkinesiae, as are also the sudden flashes of impulsive violence which occur in these patients, who will suddenly scratch, bite or strike out at a nurse or a fellow-patient, sometimes in response to some trivial annoyance, more often without visible cause. Outbursts of excitement occur in nearly all the cases, being more dramatically striking in the cataleptics: violence is almost confined to the hyperkinetic group ...

Mannerisms and attitudes. These are found almost only in the hyperkinetic group, just as in the experimental animal they occur in the

stage of hyperkinesis. Some of these patients tend to assume the "intra-uterine" position, and other strange postures. Thus one boy will sit for hours with his neck fully hyperextended; another assumes the "crucifixion" attitude; another throws back his head and extends one arm and hand in a gesture of imperious command. One patient insisted on keeping all his fingers rigidly outstretched at times, even when concentrating quite well on a Wallin's peg board during a mental test.

Self-injury. Self-injury is frequent, occurring in thirteen of the cases. Its usual forms are striking the head, tearing the skin with the nails, or biting the hand. One boy bit a large hole in his lower lip. Self-injury is, of course, not uncommon in simple profound idiocy, but there it is usually either the result of long-continued slow movements of autoerotic type or else a part of a rage reaction. In the catatonics the action is sharp and energetic, is not a response to an external stimulus, and is accompanied by varying emotional expressions; sometimes weeping, occasionally pleasure, and in two cases no emotion of any kind.

Causeless weeping. A most important sign of emotional abnormality is the occurrence of weeping or laughter without cause. This occurred in sixteen cases, and the writer has never seen it in a case which did not at the time or subsequently develop signs of psychosis. The periods vary in length and frequence, and the causeless weeping is sometimes accompanied by self-injury ...

Depth. The depth of the psychosis is not easy to estimate; the clinical picture is confused by the profound intellectual deficiency. Generally speaking, the stuporose cases are surprisingly accessible to external stimuli, and though they quickly sink back, one cannot easily distinguish between the withdrawal of stupor and the mere apathy of idiocy. The writer has only seen one case of profound stupor—the case is not included in the present series. The hyperkinetic cases are less accessible, or respond only by exhibiting further abnormal psychomotor phenomena. In both groups, and particularly in the catalepties, there is a definite day-to-day variation which is rarely seen in the stupor reactions of the intellectually normal.

A variety of clinical types were represented in Earl's series of cases:

"Simple" primary oligophrenia	10 cases
Mongolism	2 cases
Epiloia	5 cases
Acquired organic cerebral lesion	8 cases
Dementia infantilis	2 cases
Unknown	11 cases

Earl points out that because of inadequate case history data the exact age of onset of the psychosis could not reliably be determined. In the large majority of cases, however, the psychosis was already "definitely established" before the age of ten years. It is worthy of note that Earl states that in this series "no case of congenital profound idiocy occurred." In each of these cases, apparently, the original level or potential was above the level of idiocy Earl found at the time of his study.

It has been argued by some that the undisputed fact that psychotic-like, particularly schizophrenic-like, symptomatology is frequently observed in the lower grades of mental defect is no basis for assuming that psychologically we are dealing with the same condition as when these symptoms are observed in the intellectually normal person or even the high grade type of mental defect. According to this argument the schizophrenic symptomatology—particularly some of the bizarre and repetitive motor phenomena—is a more or less direct reflection of the organic pathologies which so many of these cases have. Although organic pathology certainly cannot be considered a fortuitous factor, there has been no convincing demonstration of the nature of the relation between organic pathology and the behavioral symptoms—nor does the organic pathology seem capable of explaining either the episodic or the chronic nature of the motor and emotional symptomatology.[5] It

[5] Dementia infantilis, originally described by Heller (25, 29), is a specific example of the problem, even though it is traditionally not included among the mental deficiencies. These are cases in which the child is apparently normal up until three or four years of age at which time a variety of behavioral changes are observed, the child deteriorating to a point where he is frequently indistinguishable from many "conventional" defectives of the

is our opinion, which we will more fully discuss later, that the state of our present knowledge is such that we cannot rule out the possibility that psychologic factors play a role in the psychotic-like behavior of many severely defective individuals. Until the kind of clinical investigation exemplified by Earl's work is combined with adequate life-history material—the kind of material which Earl recognized he did not have in his (institutional) cases—the question of the role of psychologic factors in the psychotic-like behavior of the severest grades of mental defect must remain an open one.

B. PERFORMANCE AND POTENTIAL

[In the full report emphasis was placed on the importance of observing the non-test as well as test behavior of the individual. It was our opinion that if we could observe the individual in other life situations, we would be better able to understand discrepancies in level of functioning between test and non-test situations and, perhaps more important, to evaluate more validly the potentialities of the individual. In those chapters we were primarily concerned with problems of the nature and measurement of intellectual processes. We feel that the same point of view may be of help here in indicating some direction in which research on personality organization and functioning of the mentally defective might take. It is our opinion that such research would be of relevance to the problem of psychosis among the mentally defective population.]

The tendency to view the defective individual from the standpoint of a test score is probably most true in the case of those labelled as idiot or imbecile. With such individuals the intelligence or developmental quotient has not only exempted them as subjects of psychological research but it has also served as an effective barrier against innovations in training and treatment. This situation is all the more surprising in view of the findings of some of the psychological research that has been done. For example, in 1931 Aldrich (2) described a study of the problem solving behavior of idiots:

> The first problem of this series involved box-stacking as a means of obtaining a lure. The child was brought to the room by the experimenter, who then remained outside, observing the child's behavior through a one-way vision screen. The lure was suspended from the ceiling, at a height proportionate to the number of boxes to be used and the height of the child. The problems increased in difficulty until they were beyond the range of the child's ability to solve them. The circumstances which accompanied success or failure were carefully recorded.
>
> The second group of problems of this series involved the use of implements. These problems were likewise arranged in order of complexity. The lure was placed in a pen six feet square, made of palings four feet high. The implements were sticks which could be used either singly or combined, according to the problem.
>
> The solutions of the problems both with boxes and sticks depended upon self-initiated activity. There were no verbal directions and no adults or other children to depend upon for cues or encouragement. The child was in the room alone, and success in the problem could only be expected when there was sufficient incentive value in the lure to bring about a spontaneous response in the child's behavior. This fact led to experimentation with the lure itself as a means of

idiot or imbecile level. Some workers reported that they found the pathologic anatomic basis of this condition and therefore excluded them from the schizophrenias. Others could not corroborate these pathologic findings and considered it a variety of schizophrenia. As with the severe grades of mental deficiency, the inaccessibility of these cases makes the usual psychologic and psychiatric diagnostic modes of study almost impossible. It is our opinion that it is unlikely that a specific pathologic brain condition will explain the complex behavioral changes described in these cases. This is not to say that brain pathology may not be an etiologic factor but rather that it interacts with psychologic factors which also play a determining role in the course of the condition. We would agree with Earl's (15) conclusion about the etiology of psychosis in idiots: "The etiology of the condition is a subject for further study. The present evidence would appear to indicate the causal importance of physical rather than experiential factors. The final decision cannot be reached until much more is known, both of the behaviour patterns of idiocy and of their physical correlates."

studying the effect of incentive on the individual performances.

The first lure was a ball, which in no case failed to attract the child's attention and bring about at least a reaching response, the most primitive form of adaptive behavior in such situations. Then a ball and a cookie were presented, and finally a banana.

As judged by the amount of activity induced in the subjects and the number of successful responses, the ball, although an eagerly accepted toy, held less incentive value than the cookie, while the banana surpassed either of these. Tommy, for example, piled two boxes together for the ball and cookie, but absolutely ignored the third box. However, when a banana was substituted as the lure he immediately stacked three boxes together. Such an observation leads to the speculation that ability cannot be determined until stimulation is maximal.

Billy was obviously afraid of standing on three boxes after he had stacked them together. He attempted it several times when the ball was suspended above his head, and each time gave up. When the banana was used, however, he immediately stood on the same three-box structure, displaying no overt fear reactions. Incentive in this observation seemed definitely associated with the elimination of an emotional obstacle to success.

Many responses in the situations revealed personality differences of much importance. For example, when the child was taken to the room and the door shut between him and the experimenter, there were many kinds of reactions. One child cried at being left alone, and he continued to do so until it was necessary to take him from the room. Others whimpered when their attempts failed, and still others showed evidence of discontent and anger under such circumstances by stamping or banging on the door with their fists. However, the latter displays accompanied failure and not mere discontent at being alone.

The reactions of the children during the momentary presence of the experimenter at the beginning and end of the trial also suggested personality differences.

For example, Sammy began his solution of the problem the minute the door was open for him. Carl waited until the door was closed, with the experimenter outside, while Jimmy waited until the end of the trial period, when the experimenter had re-entered the room, before beginning any activity toward the ball, and then looked constantly for approval and assurance.

These personality differences displayed according to the presence or absence of the observer, suggest important considerations with respect to adult presence and attention in a training program. Obviously we must treat every child individually, encouraging those who respond best to encouragement and isolating those who develop best in self-initiated activities.

Distractibility was sometimes prevalent and in some cases associated with failure. George, who has very low intelligence and meager adaptability, was hyperactive upon seeing the ball, and reached for it repeatedly. But he did not use the boxes at all until the experimenter placed them directly under the ball. George then became engrossed in climbing up and down, and completely ignored the ball. In this case the solution itself was a distraction in obtaining the end result.

Binet mental age within the limits of the group was not a good measure of a child's ability to solve these problems. The three who failed the simplest problems were of the lowest mental ages. However, the seven subjects who ranged in mental age from two to three years did not seem to be sufficiently differentiated by the tests to account for their varied ability in these situations.

These and similar observations of the idiot's activity in problem situations at the borderline of his ability suggested incentive, emotional makeup and individual personality reactions as well as mental age as influential factors in idiot behavior and trainability.

Although Aldrich employed a "test" situation, it obviously differed in important respects from the formal testing situation; in fact, level of performance between the two situations was not highly correlated. Although the following statements are contained in the above quotation, we think they are deserving of restatement:

1. Under conditions of strong motivation, some severely defective individuals are capable of directing and sustaining their attention to the external environment, their level of problem solving in the process being surprisingly good. If we knew more about the

everyday behavior of these individuals, this conclusion might take on added significance.

2. From Aldrich's description it would seem that some of these individuals were responding in more than a concrete fashion. One hesitates to ascribe to these individuals the capacity to reflect and organize but it seems to us difficult to square the descriptions with the assumption that these cases could respond *only* in a concrete, passive, unreflective way.

3. There are marked personality differences among severely defective individuals —suggesting that the kinds of interpersonal relationships which they have experienced were not identical. Put in another way: the factors to which we ordinarily attribute importance as a source of individual differences may be operative even when dealing with severely defective organisms.

There are a number of grounds on which one might question these conclusions. One might say, especially in regard to the second conclusion, that these cases were not "true" idiots but individuals who had in effect regressed from a higher level of functioning. But what are the personality dynamics and external conditions associated with such regression? If one invokes some organic pathology, how is one to understand the resulting individual differences? One need not resort to a construct of regression, embedded as it is in a theory of personality, and simply state that some of these cases were just not given an opportunity to develop what capacities they had; they were given a label and then handled in a manner considered appropriate to it. This is in effect saying that some of these individuals suffered from *understimulation*, an argument rarely advanced with the severest grades of mental deficiency. Firm conclusions from Aldrich's study cannot be drawn, although similar findings have been reported by Gardner (20).[6] The point that seems least questionable is that even in the severe grades of mental deficiency individual differences *in behavior and personality* exist. This is but another way of saying that we are dealing with individuals whose behavior is not due to static factors but to a dynamic interaction between internal and external forces which reflect previous experiences, in short, these dynamic interactions have a history. From this viewpoint it should not be surprising, therefore, that personality changes over times should be noted in some of these individuals, even some of the marked and dramatic changes noted previously. The research problem is not so much what we label some of these changes (*e.g.*, psychosis) but in understanding their psychological basis and implications for training and treatment.

SIGNIFICANCE OF ITARD'S STUDY

It is something of a commentary on our contemporary knowledge that the best description yet made of the behavior of a severely defective individual is that by Itard (30) on the Wild Boy of Aveyron. For the purposes of our present discussion the fol-

[6] In 1948 McPherson (43) presented a comprehensive review of experimental studies of learning in subnormal individuals, several of the studies being concerned with the severely defective cases. It is not surprising that McPherson concluded: "The outstanding impression gained from this review of learning in the subnormal is one of lack of information. The actual experiments have been few, the number of subjects small, the tasks to be learned heterogeneous within a narrow range, and the motivational factors inadequately controlled. The results of this review serve not so much as an aid to the technician in meeting clinical problems but as a reminder to the experimentalist." However, the following conclusions did seem to be supported by findings of more than one investigator: (a) changes in incentives or motivation resulted in increased learning and (b) there is a lack of relationship between learning behavior and intelligence test score.

Recent studies in England by Clarke and Hermelin (14), Gordon, O'Connor, and Tizard (21, 22), Loos and Tizard (40), and Tizard and Loos (55) certainly are in line with the conclusions and findings given above. Clarke and Hermelin concluded: "It seems that the limits to the trainability of imbeciles are very much higher than have been accepted traditionally either in theory or in practice." A recent book by O'Connor and Tizard (44) has summarized most of the English studies.

lowing should be said of this classic:

1. At the time that Itard took Victor, the Wild Boy, under his wing there was no reason to believe that the boy had ever functioned at a higher level. Itard was viewed as something of a fool for attempting to work with the boy. We make this point because we think it not unlikely that were someone today to give as much time to an idiot or imbecile child as Itard gave to Victor, he probably would be viewed not as a fool but as someone with a curious penchant for wasting time.

2. Although Itard was disappointed that he could not make Victor into a normal youngster, what he did accomplish with the boy can be appropriately labelled as phenomenal. Over the long period of training one could see the development of various ego functions, the capacity to delay responsiveness—in short, one saw the development of a surprisingly complex personality.

3. Perhaps the most important conclusion that could be drawn from Itard's work is that even in severely defective individuals the quantity and quality of interpersonal relationships is an important variable in determining the level of complexity and efficiency of psychological functioning. Put in another way, even in the severely defective child the measurement of intellectual performance and potential cannot be meaningfully done without consideration of environmental opportunity and stimulation.

SIGNIFICANCE OF RESEARCH WITH THE SEVERELY DEFECTIVE INDIVIDUAL

Thus far in our discussion we have concentrated on the severely defective individuals because, in addition to the research implications already noted, there are other considerations which make psychologic research with these cases important. For example, it has been noted by Lemkau (39) and others that the distribution of intelligence may not be a normal one because the number of idiots and imbeciles is apparently significantly greater than one would expect from the normal probability curve. On the basis of our above discussion we think it not unlikely that such a conclusion may be a function of the practice of using test scores which reflect performance rather than potential.[7] If in some of these cases we are dealing with personality regression, in others organic deterioration, while in others inadequate training and stimulation have not allowed for full development of potential—if these conditions occur among severely defective individuals, statistics about the incidence of idiots and imbeciles (as identified by test scores) cannot be considered as being very meaningful.

As soon as one seriously considers the possibility that even in severely defective individuals the quantity and quality of interpersonal relationships are important variables in development, the role of parent-child relationships must be evaluated. In recent years we have become aware of how the severely defective child affects the lives of parents and siblings but little or no attention has been given to the influence of parent upon child. In fact, when one considers the psychologic disruption which frequently occurs in families with a severely defective child, it is difficult to avoid the conclusion that the child in some way is affected by it. That this possibility has been little discussed, let alone studied, is probably due to the implicit assumption held by most professional workers that the severely defective organism has no personality or intellectual potential to speak of and therefore one need not be concerned about psychologic consequences. We have no doubt that in some cases such an assumption is warranted, but we also have no doubt that in some cases the assumption is without foundation.

The following question may have occurred to some readers of this report: assuming that what has been said so far has some merit,

[7] It is interesting to note in this connection that Earl (15) concluded that none of his psychotic cases of the idiot level were "congenitally profound idiots" as their behavior at the time of the study might have suggested.

how can one justify the expenditure of time, money, and personnel for research with individuals who will always be in need of the closest supervision, even in the institutional setting? This question can be answered in several ways. On the most practical level one could say that to the extent that such research increases our psychologic understanding of these individuals the problems of care and training would be correspondingly decreased. If such research would serve to enable us better to help parents in the handling of these children, it would represent an important social contribution. Again on a practical level: although the knowledge we may gain from such research may never enable us to help the severely defective individual as much as one would like, it may be of great value when applied to the less severely defective individual. If by our increased understanding of the severely defective individual we can learn how to modify his behavior and increase his range or adequate responsiveness—however small such modifications and increases might be—then we would have reason to expect that similar endeavors with the less severely defective individual would be more productive.

THE PROBLEM AND NEED OF THEORY

Perhaps the greatest significance of research with the severely defective individual is the challenge it presents to psychologic theory and practice. How adequate are our psychologic theories for understanding the problems we have thus far discussed? The fact that most psychologic theorists have never been concerned with problems of subnormal functioning is less important than the fact that we cannot assume that their theories would at all be adequate to cope with these problems. By adequate we mean that a meaningful research program, derived from a systematic theory, could be set up which would show promise of illuminating the kinds of problems we have been discussing. It should not be overlooked that the idiot and imbecile are human beings, and any theory which purports to be a theory of human behavior should be able, if not to give satisfactory answers to the problems which the behavior of these individuals present, *at least to indicate how to go about finding the answers.* The problem of theory is basic because we all operate, implicitly or explicitly on the basis of some theory of human behavior. It is only through the process of scientifically *testing* our theory that we can evaluate its adequacy as a basis for our practice. It is because psychologic practice must be based on some theoretic conceptions that concern with psychologic theory cannot be viewed as a luxury.

To our knowledge there has been but one attempt to view mental deficiency (particularly of the severe grades) from a systematic, theoretical position. We refer here to Clark's (10–13) attempt to view the problem from the standpoint of psychoanalytic theory. There is much one can question and disagree with in Clark's ultraorthodox psychoanalytic formulations. Aside from the fact that he was employing analytic theory of the 1925 variety, the major difficulty one encounters in reading Clark is his completely uncritical acceptance of the validity of the theory in all its aspects. The quotation which follows is less open to these weaknesses, and can serve to illustrate some of the points we have attempted to make: (12, p. 36)

> ... if we say that mental deficiency consists of some failure in the processes of acquiring, absorbing, and using knowledge for an adaptive mastery of reality, what are the specific defects which lie behind this failure? In many instances, brain lesions and organic injuries or defects have been indicated as the basis for a mental arrest. Other investigators have referred to "a pathologic variation in the germ cell," which makes complete mental growth impossible. Another plausible explanation for mental deficiency is advanced by those who hold that there is a defect or arrest in the development of the neurons. Each of these points of view would seem a reasonable attempt to determine the basic cause of mental arrest. Each consists of a formulation which the observed data frequently appear to

warrant. Yet we feel that none of these theories can indicate just how the fundamental cause leads to the difficulties which the ament is seen to have. Nor do they contain possibilities for understanding the individual in such a way as to help him in his problem of getting along in the world.

Our belief is that even in cases where a definite organic injury is present, there are dynamic or psychologic factors which play a prominent part. The ego, as we view it, is not only an organization of character patterns and abstract capacities, but also is made up of the sum of the physical elements constituting the human body. The total "self" would consist of the individual's body, his mental impressions from it, and his co-ordination of the somatic, as well as the psychic elements referring to it. Any wound to the psychical structure must be reflected in the ego's efficiency and in its sense of power to govern the total organism in its approach to reality. Especially must a handicap result, if the injury is to that portion of the machine which is most sensitive and most needed in the process of guiding or directing. A lesion in the brain, therefore, or an injury to the central nervous system would greatly impoverish the weapons by means of which the ego carries out its appointed tasks. Furthermore, a psysical injury of this nature results in a severe psychic wounding of the ego. The sense of selfassurance and confidence is deeply hurt, for the ego misses some part of its feeling of completeness. In a sense it has been castrated, and it automatically seeks some emotional compensation. Case-studies have suggested that the ego's need for love is greatly emphasized in such instances; that there is an added impounding of libido within the personality, as a narcissistic agency for psychic healing and soothing. An injury coming early in life might thus serve to inbind a more solid formation of primary narcissism, and the extra narcissistic need might well inhibit the full ingestion and absorption of identifications. In other words, a lesion might cripple mental development, not directly, but through its burden of handicap upon the ego and the libinal processes necessary for mental growth and functioning. We must grant to organic factors, wherever present, a share in the causation of mental enfeeblement; but we hold that it is mainly through its effect upon the ego and upon the distribution of available energies that a physical defect exerts its mental crippling. If we regard the problem in this way, it seems to bridge the gap between an atypical organic "cause" and a "result" in the realm of mental functioning...

We should prefer to summarize these basic crippling factors and recognize them simply as constituting a fundamental ego-defect. Whether this be in the germ cell or in the development of the neurons, we may at least say that the total ego is without some degree of its usual endowment for meeting reality. In dealing with amentia, whatever more remote etiologic data may be held responsible for its inception, our major concern practically is with the imperfections noted in the ego's functioning. Here we must leave concrete, physical representations and turn towards capacities which have to do with definite mental processes. Our point of view is that the ego's defect lies within such capacities as perception, memory, ideation, judgment, and reasoning. These are specific phases of its endowment, necessary for mental grasp and adaptation or mastery of its surroundings. They may be originally handicapped from some physical lack in the elements which go to make up the ego (germ plasm, neurons, etc.); they may be crippled at some time as a result of organic injury (lesions, etc.); or they may be innately defective themselves as capacities forming part of the patterns which the ego inherits. Whatever the ultimate, basic cause, the important fact is that these capacities are the defective elements most typically observed; and these functions are the ones which need helpful understanding if development is to be brought up to the highest possible level of individual usefulness and happiness.

Our opposition, therefore, is not against the scientific investigators who indicate organic or constitutional defects as the basis of mental arrest. Rather, we take issue with those who infer from these findings that "therefore nothing can be done about it." Our opinion is that a recognition of certain unchangeable, irremediable factors in an individual is absolutely necessary, but that within the limits imposed by such physical or deeply ingrained handicaps there is much that can be better understood and more constructively dealt with.

It is for this reason that we emphasize the dynamic elements in the problems of amentia. They are the moving forces of energy which must be used in the actual functioning of whatever

capacities the ego does possess. They are the vitalizing powers which are necessary for advancing mental development as far as inherently possible, and for directing this development into the channels of greatest individual efficiency. We refer specifically to instinctual energy, for it is from the libido and from the destructive impulses that the driving force for all contacts with reality seems to come.

Thus the main, immediate problem in mental arrest seems to concern the aspects of ego-development and the uses to which emotional energy is put. If we are to understand and help retarded individuals, we need to study the formation of primary narcissism and its effects upon the capacity for making identifications. It will be pertinent to investigate the manner in which oral-libido is used with the destructive impulses for an ingestion of knowledge from the outer world. We must get a clearer insight into the development of secondary narcissism, its advantages in supplying the urge towards grasping reality, and its disadvantages in obstructing the full absorption of what is taken in. Our understanding of the ament will be further advanced if we can observe the extent to which he has attained objectivity, by means of which he can reproject and use his acquirements for adaptive purposes. And finally, we may better appreciate his difficulties in attaining a social adaptability and in adhering consistently to moral codes when we see more deeply into the formation of the super-ego.

These are the factors we feel are all-important in any theory of mental arrest which strives to deal with backward individuals as living beings. They are the elements which seem to influence the ament's functioning in everyday life and his possibilities for a restricted but at least a further elaborated development.

The above quotation can serve as an example of how theory can direct our attention to problems ordinarily unnoticed. An important problem in analytic theory concerns the process whereby the dependent infant, whose behavior appears almost exclusively determined by *his* experience of pleasure and pain, becomes capable of directing attention and interest to another person and responding in a way so as to obtain "pleasure" from that person—the transition from a state of primary to secondary narcissism. It is assumed that this transition takes place by means of a process of identification with an external figure and that this process already reflects certain intact ego functions (*e.g.*, perception, memory, increased control of motility). When these ego functions are defective in the very young child, it becomes much more difficult, if not impossible in some cases, to make the transition from a state of primary to secondary narcissism. What is important in Clark's formulation is that he raises the possibility that the defective ego functions may have the effect of reinforcing or fixating behavior on the primary narcissistic level, and that an *unusual* amount of attention and understanding would be necessary if the child were to begin to make the transition. From this theoretic formulation, therefore, research interest becomes focussed on the psycholigic development of such children and the external conditions necessary for facilitating maximal development of potential.[8] Put in another way one could say that this formulation suggests that the behavior we observe in these cases, even in the severely defective ones, may not be representative of what they could do under other conditions of rearing and training. It is not surprising that Clark makes special reference to Itard's work and attributes the changes observed in Victor to the unusual amount of attention, love, and stimulation which Itard and the governess gave to him. It should also be noted that in the two severely defective boys whom Clark described, and with whom a therapeutic approach was attempted, support for the above formulation is found. In these two cases, however, the amount and quality of attention given does not begin to compare with that described by Itard.

[8] Despite his formulations Clark, strangely enough, never gives what we would call a case history for any of the children he describes. Nothing is said about the parents or problems and methods in rearing the child, nothing about the earliest phases of development which are so crucial in his theory.

There are several things which should be emphasized about Clark's theoretic formulations in order to illustrate the fruitfulness of viewing specific problems from the viewpoint of a general, systematic theory. First, it directs our attention to the earliest phases of development and emphasizes the importance of special handling *at those times.* It also makes us aware of how little interest has been shown in describing and understanding the ways in which the defective infant or very young child is reacted to and stimulated by parents.[9] Second, the theoretic formulation suggests a course of action. The process of testing the theoretic formulation involves attempts to change the behavior of the patient. In the work of Clark, as well as that of other analytic work (1, 9) with defectives, the course of action has basically involved the establishment of an interpersonal relationship and the subsequent use of this relationship to increase the range and adequacy of the individual's externally directed behavior. Even with the severely defective individual, as described by Clark and Itard, this type of relationship can frequently be established.

Following the completion of this section an article by Benoit (3) appeared which deserves mention because it is one of those rare efforts to discern the practical significances of a systematic theoretical position. In this instance, it was Hebb's (27) neurophysiologic theory of brain function which was employed. The present writers are not competent to evaluate either Hebb's theory or the manner in which it is employed by Benoit. However, there is no doubt in our minds that the problems to which Benoit's attention has been drawn are important and one can only hope that they give rise to a formal research program.

1. Hebb's theory has been concerned (among other things) with the problem of the adverse effects of stimulation privation in early life.

It seems appropriate to inquire into how the theory might illuminate and make possible a more effective control over the learning process in the mentally retarded. For one thing, there is a tendency for parental attention to shift to some extent from training in self-help and other activities to excessive care when reactions and general behavior are seen to deviate from normalcy; and there arises the question as to whether this inclination to over-protection might not have a destructive or stunting effect on the development of the child, over and above any neurological impairment that may occur. Furthermore, because the evidence gathered from animal studies seem to support the claim that stimulation privation in early life produces adverse effects even in late maturity, one is prone to wonder whether a conscious effort to lay more emphasis on training with reference to both amount and degree of adaptivity might not result in raising the achievement level in all the major spheres of performance (3, p. 500).

Benoit's focus on parental handling of the subnormal infant, and the possibility of understimulation in these instances, coincides with the speculations of the writers' earlier in this section.

[9] This statement may not adequately convey the complexity of the problem. For example, there is probably great variation in when the diagnosis of severe mental deficiency is made or communicated to parents, so that these children differ in the amount of time they were responded to as normal children. We do not think one can assume, without further evidence, that to be handled and reacted to like a normal child can only be "good" for the severely defective child. In addition we do not know what changes take place in rearing practices after the parents know of the child's condition. We think it would be more than worthwhile if studies could be made of (a) the *specific* advice which parents are given in relation to ways of handling the infant or young child, (b) the justification which the physician can give for such advice, (c) and the adequacy of such advice when viewed in light of the every day problems which such children present. It is our impression, which one of us (51) has elaborated on elsewhere, that the very nature of the diagnosis, the way in which it is frequently communicated to parents, and the vague or too general advice given to parents about details of rearing the child often produce marked psychologic disruption in the parents. It would be doing violence to any psychologic theory of child development to assume that the child, even the severely defective, was not in some way affected by such disruption.

2. "The key element in the learning process is attention. In the Hebbian framework, the control of attention is assumed to be achieved through the delivery of facilitation from one organizational structure to another. Accordingly, in order to ensure attention, structure that may logically be presumed to be appropriate antecedents to any given response must be activated; that is, a set must be established." Benoit points out that the establishment of appropriate sets in the defective organism may be made impossible or interfered with because of that organism's incapacity to handle or organize the variety of stimuli which impinge upon him. "One may infer that the successful production of new learning may require the elimination of irrelevant stimulation. When learning is very imperfect a complex situation may elicit mass activation, which obviously results in diffuse thinking. Accordingly, the theory suggests that the teaching situation be made more effective by simplifying contexts. By diminishing the volume of stimulation, there is less chance of disturbing a given organizational structure while it is in process of being reinforced with a repetitional series." This conclusion is very similar to that of Strauss and Kephart (54). Both Benoit, as well as Strauss and Kephart, discuss the problem of control of irrelevant stimulation primarily in terms of the older, defective child. It would be our suggestion that equally (or more) important is the study of this problem in infancy and very early childhood, not only because they are periods of initial learning but also because they are the times when the parents of the defective child receive the least guidance in training procedures. The significance of these early periods is central to Hebb's theory as the following statement by Benoit indicates:

> Finally, Hebb strongly urges that active concern with intellectual training begin early. On the face of it, a new organizational structure can be formed at any time in the life of the individual, provided the necessary maturational level has been achieved. Actually, however, the situation is not so simple. As a person ages without opportunity for new experiences, without acquiring new knowledge or new organizational structures, learning sets weaken or become relatively ineffectual, owing to the reversibility of the growth process that gives rise to the structures that mediate learning; in other words, behavior becomes permanently constrained within the limits of primitive habits. There is considerable evidence to the effect that providing the young with abundant suitable environmental stimulation greatly affects the course of their development right on into adulthood, whereas the same advantage later in life produces much less startling results.

In our opinion, Benoit's article not only underlines the importance of theory as a guide to problems and practice but also emphasizes the need for closer study of the experiences and training of the defective child in his earliest years.

BOURNE'S PROTOPHRENIA

In 1955 Bourne (6) reported a study of severely defective individuals which because of its implications requires special mention. His subjects, all with I.Q.'s below 50, were 154 young children admitted consecutively to an English hospital.

> Of the 154 cases, 138 had an evident organic cause and 16 had none. The distinction was based on clinical investigation and a medical history derived from several sources. The chief source was standard history forms completed by the hospital's social workers in each case, and in 74 by the outpatient consultant as well. Also there were 330 medical reports—an average of 2 per patient in the larger group and 3 in the smaller—specially requested from hospitals attended previously. Many of these were really plural reports, concerning more than one admission, or coming from independent departments, of referring to other members of the family too; in 100 of the cases, including 14 of the smalled group, the pregnancy and labour were described. In 34 cases I interviewed relatives, mostly at their wish, but in a few cases to get extra details.

There were 36 deaths, all in the larger group, 31 coming to necropsy, and in all of these organic disease was found.

...At the Fountain Hospital, with 600 beds for mentally defective children, almost all under 5 years old on admission, the processes causing severe mental defect are relatively recent and thus unusually accessible. It will be shown that clinically, as well as at necropsy, most of the severe cases can be explained by organic brain disease, and that heredity seems much less significant than is sometimes believed. There remains a small group of children, lacking any apparent organic or familial pathology, who appeared to me to display notably both a curious pattern of abnormal behavior and a background of appalling misfortune. It was postulated that in such cases a faulty upbringing might cause failure of mental growth.

...The information was gathered from the sources mentioned for the medical history, the standard forms ensuring certain items uniformly for all cases, and the reports from other hospitals supplying occasional psychiatric accounts of near relatives; in addition, there were 209 reports from outside social agencies, mainly local-authority workers.

Where occasional discrepancies were met, an obviously less reliable report was discarded, or further inquiry was made. My judgement replaced that of others in a single instance:

All accounts referred to a mother as mentally defective because she had been certified as such in prison and spent years in a colony. As her letters were far too fluent for this diagnosis, she was interviewed. She proved to be a psychopath, of at least average vocabulary and literacy, from a criminal family, and now the prosperous young widow of an engineer who had died "suddenly." If she was defective, it was in morals and not intelligence.

For statistical treatment, the social histories compiled in this way were tabulated, the items extracted for this falling into some fifty categories. The two groups were then compared statistically, item for item, mainly by the χ^2 test with Fisher-Yates correction, required because one group was numerically small.

Because the number of mongols in the organically affected group was large, it was necessary to prevent unknown factors that might be peculiar to them from influencing the comparisons, either by swamping real differences or by creating misleading ones. As a precaution, every calculation had to be repeated with mongols treated as an isolated group. In the event, this turned out to have been superfluous, uncovering nothing otherwise overlooked, and indicating only the differences to be expected between mongols and the rest of the organic cases.

We shall not go into the details of Bourne's findings. Suffice it to say that his data indicate that the small group with no organic pathology came from backgrounds which, both before and after their birth, could be described as psychologically unhealthy in the extreme.

The outlines of the condition isolated now emerge. The young child, though physically healthy, will present severe backwardness evident from about the second year, with curious behaviour disorder and a history of perverted mothering in infancy. In his first two years he will either have been reared by an extremely disordered person, commonly a psychopath, or else deprived for long and repeated periods of the mother or her substitute; usually he will have suffered both misfortunes. None here were simply reared in an institutional setting: of 3 so reared from the age of 18 months or less 2 first endured terrible neglect by unstable mothers, 1 passing later through five institutions, while the 3rd went from foster-mother to institution to institution.

Clinically these children's backwardness is uneven; despite an I.Q. about 40, their early milestones are not much delayed, and they lack the clumsiness and impoverished expression of other imbeciles—some, indeed, are very graceful and attractive. At times they appear extraordinarily remote and even deaf, tending to monotonous mannerisms, banging their heads, and tearing their hair out; many also have intolerable propensities of screaming and destructiveness.

Of those I have observed in hospital for a year or two beyond their fifth birthday, some became docile imbeciles, and others burnt out and dilapidated ones; yet others do not change.

The condition described here must be of great social interest since it cannot be rare, it is probably preventable, and it may initially be treatable.

At the Fountain Hospital about 10 per cent of severe aments seem to be in this category. Admissions are from a big area and without any special selection, and this figure must be fairly representative of extreme defectives under hospital care. The condition may therefore be responsible for many thousands of people occupying hospital beds in this country. Moreover, this is a restricted estimate confined to psychogenic defect in apparently pure culture. The condition may be even commoner for the following reasons: (1) Milder emotional undernourishment or damage, or an equal degree in a more resistant victim, may account for some of the less extreme defectives in the community, and even more of those in institutions. (2) Some mental defectives of organic aetiology may have been reduced to the institutional level by such trauma, additionally. Possibly these children are less equipped to overcome indifferent rearing and very liable, by virtue of physical deformity, to elicit it.

The histories of these cases monotonously disclose how little realisation still exists among social agencies, doctors, and hospitals that a small infant is a social being who needs more than food alone to grow up. For example, the digestive difficulties, failure to thrive, and napkin rashes common in these infants, and a crying illustration of maternal incompetence, led to their spending weeks at a time in hospital, always to be discharged "recovered," and always to relapse with another complaint, but never led to serious probing of the mother. Even when backwardness was obvious, the exclusion, often recondite, of physical disease left inquiry exhausted; in all five cases in this series where the child was under one hospital while the mother was under another for psychological treatment no communication appears ever to have passed between the two.

... It will have become apparent that these children may superficially resemble adults with chronic schizophrenia. However, similar behaviour does not mean identical psychopathology, and merely to label these defectives as psychotic deprives the term of any precise meaning. Generally, psychosis designates disorders where ego functions have no such disorder, and far from presenting disintegration, with two exceptions, they achieved integration at no time at all, their disruption being presumably of primitive infantile processes assembling towards an organized personality. The early distortion of those unintegrated processes that survive may account for the curious behaviour which characterises these patients.

Protophrenia seems to me a suitable name for this condition, implying an aborted organization of the personality as distinct from disorganization, to which psychosis refers, and from undistorted retardation accompanying organic cerebral defect, which, by itself, seldom produces autistic behaviour disorder, to judge from the material here.

It is uncertain how protophrenia is related to the syndrome of "early infantile autism." There seem to be differences in that a proportion of the cases here are not autistic and in that only some of Kanner's cases were regarded as mentally defective and none are described as having behaviour disturbances apart from lack of emotional relationship to people and monotonous adherence to some daily routine. The background of his cases was different, too, containing an excess of socially and intellectually superior forebears.

Protophrenia can be regarded as a psychogenic failure of ego formation. Its study must be relevant to that of pathological ago formation, generally, where early emotional trauma is at all important. This includes the fragility underlying adult schizophrenia and child psychoses, and the maldevelopment of psychopathic adults and problem children. The present arrangement whereby mental deficiency, child guidance, and adult psychiatry are practised in almost complete isolation from one another can only impede understanding of the common processes basic to each.

As Bourne recognized, the number of cases is small and the study requires replication by others. In such replications it would be recommended that the reliability of clinical judgement concerning organic pathology or not pathology be determined in order to evaluate unwitting bias due to knowledge of case history data. In his study Bourne did both the clinical examinations and analysis of background data. Bourne was aware of this source of contamination of judgement and seemed studiously to try to avoid its effects. But the methodologic prob-

lem remains and should be sequarely met in future studies. We would agree with Bourne that his findings are potentially of great significance and demand further study.

An important question raised by this study may be put as follows: why is it that some children who have been subjected to the kinds of experiences and relationships which Bourne describes do *not* develop the picture of severe subnormality he calls protophrenia? It probably would not be difficult to find children who have been subjected to even worse experiences and relationships but who do not manifest such an extreme picture. We are here raising the possibility that there is a predisposing factor which selectively determines the degree to which a child will be affected by certain untoward experiences or relationships. We shall return to a consideration of this problem following a discussion of the idiot savant and infantile autism. At this point we wish merely to state that while it has been obvious that there are temperamental and behavioral differences among children from the earliest days of life, and it has been assumed that such variations are important in some way in atypical development, the problem has been little studied. As we hope to indicate later, however, certain clinical observations reported in the past decade or so allow one to think more specifically about the nature and varieties of predisposing factors.

THE IDIOT SAVANT AND INFANTILE AUTISM

Although the idiot savant has been described and discussed many times over the years, it was not until 1945 that a really penetrating and comprehensive review and discussion of the problem appeared. In that year Scheerer, Rothmann and Goldstein (52) published their monograph, a work which has not received the attention it merits. The reader is urged to read this monograph from which we give below the authors' summary statement:

L., an 11-year-old boy, with behavioral peculiarities, has been studied over a period of five years and a reliable record of his previous development obtained. L. showed distinct musical aptitude—he played melodies on the piano by ear—and was remarkable in verbal retentiveness. His skill in rapidly manipulating simple numbers was also unusual, and he performed so-called calendar calculations amazingly well. Numbers he remembered with the same ease as occurrences which to his mind had once become connected with them, so that he volunteered dates, names, places and times of events at the slightest provocation.

In spite of all this, L.'s general information was surprisingly subnormal, and, with the exception of the just mentioned aspects of his surrounding, nothing aroused his interest. He never absorbed or learned in a normal fashion, nor could he attend a regular school. He was retarded in the mastery of many skills, commensurate to his age, and he was lacking in social awareness with a limited repertoire of social responses. L. had an I.Q. of 50 which classifies him technically an "idiot-savant."

An investigation of L.'s personality structure was carried out with specially devised experiments, with standard tests and with careful exploration of his spontaneous behavior in every day life. An analysis of all data and of the findings in multiform performance fields failed to disclose an individual segmental defect or several specific defects. Instead, his various deficiency symptoms pointed to a functionally common disturbance, a general impairment of abstract capacity (*e.g.*, in the semantic use and ideational understanding of language, of social contents and relations; in reasoning, in the grasp of causation, of logical meaning, of symbols, of conceptual number relations; in the cognitive structuring of visual performances and visuomotor tasks). This picture of general abstract impairment was corroborated by the experimental evidence that L. succeeded in his own performance-specialties without having a genuine understanding of their meaning as to content and implication. Further exploration of his thinking, learning and social behavior revealed an abnormal concretization. He could only grasp and learn what made situational or tangibly patterned sense to him. Otherwise, if he retained at all, it was in an automatic, associative manner by habituation.

A positive evaluation of his successful per-

formances, abilities and skills was attempted. After having experimentally ruled out other alternatives, it was found that L. possesses an initial endowment in the acoustic and audio-motor sphere, probably supported by kindred imagery. This endowment expressed itself particularly in his sensory motor receptiveness for melodies, *i.e.*, for acoustic "Gestalten" and for verbal patterns. On this basis his musical performances, his verbal and tonal memory (absolute pitch), and his aptitude for serial grouping became explicable (*e.g.*, his rapid oral counting and spelling, forward and backward, his large diget span and his calendar performance).

Further analysis of his procedure in the utilization of this endowment revealed, however, that it did not operate in a normal manner. It was bound to an abnormally rigid concreteness and functioned in a sterile, bizarre and undiscerning form. Symptomatically, in music he could not develop his talent through study or practice. His performances were desultory, depending on specific circumstances and his interest ranged from obsession with a special phonograph record to appreciative enjoyment of opera arias or Handel's "Largo" on records and indifference to *any* radio music. Correspondingly anomalous function of his talent was manifest in his excessive tendency to count indiscriminately, and to resort to enumerative verbalization and inane speech cliches whenever he was confronted with a task that overtaxed his power.

We find then that an individual who is handicapped in abstraction and endowed in a particular field or performance shows a *sub*normal intelligence and an *ab*normal canalization of his endowment. In interpreting this personality picture we concluded that owing to his impaired abstract attitude, L. cannot realize his remaining potentialities in a normally integrated manner. He is therefore driven in an abnormal degree and direction to exercise those functions which nature permits him to develop, because these are the only performances through which he can actualize himself and come to terms with his surroundings. The least impaired function thus becomes a coping mechanism of adjustment, but, since it can only operate on the level of concrete reactions, it becomes canalized into atypical forms of expression. This result seems to point to an organizational interdependence of basic psychologic functions. Certain pertinent implications with regard to normal personality structure and with regard to the problem of idiot savant are discussed and the following generalizations considered:

1. How an endowment operates and develops, depends upon the organization of the person as-a-whole.

2. There exists a functional interrelation between abstract capacity, intelligence and special endowment.

a. The abstract capacity is essential for the normal functioning of intelligence. The bearing of this on the association theory of intelligence is followed up.

b. The abstract capacity is conditional for the normal functioning and development of an endowment, although the latter may be anchored in concrete processes, *e.g.*, of Gestalt type.

3. The term idiot savant is a misnomer. Idiot savants are talented aments who possess an amented talent.

In order to test these conclusions a comparative study of other reported cases of idiot savant was undertaken and various explanatory hypotheses of other authors were critically examined. The evaluation of this case material seems to confirm our interpretation, since no cases were found in which a talent functioned normally in an individual with abstract impairment. These led to the establishment of certain criteria for the psychologic identification of a talented ament.

In studying the reported superior abilities in aments two phenomena invited particular attention because they posed an intriguing psychological problem. These were (1) the relative frequency of number manipulation and retention, and (2) unusual features of memory.

1. The analysis of the psychologic processes involved in these number performances laid bare their origin in concrete perceptual counting procedures with specific limitations. A comparison between this stage of primitive, concrete grouping and the initial procedures in arithmetical prodigies showed basically common characteristics. It became clear, however, that the arithmetical prodigy who developed further towards a cognitive understanding of mathematics outgrew this original stage of concrete dependency is his number operations, because he could increasingly adopt an abstract approach. In con-

trast the abstract impairment prevented the talented ament from passing beyond the initial stage of concrete grouping procedures.

(2) The striking retention of numbers or of outlandish and irrelevant data, as, *e.g.*, railroad tables or an entire newspaper column, is all the more surprising as in most every instance the retained material is not understood by the ament in a normal way. A psychologic appraisal of the changes in figure-ground organization as experienced during pathological concreteness led us to set forth a new explanation of these peculiar memory processes. The observations on the cases studied indicate that a defective organism will cling tenaciously to those aspects of a situation and those features of material which makes concrete palpable sense to him, *i.e.*, with which he can deal successfully. These aspects are thrust into the foreground of the phenomenal organization as the "figure." Such a difference in perceptive centering in the abnormal's coming to terms with the world of the "normal" leads to a different centering of performance. Therefore, these aments retain easily what may appear senseless or peripheral or irrelevant to the normal observer. To the aments in question, however, this is the only "sense" possible and pivotal in the experienced contents. This explanatory attempt is tested on diversified case material. Finally, comparing abnormal concreteness in aments with stages of concrete reaction in normal children the following question is raised: Does the atypical memory organization in a subnormal child represent a lawful modification of a normal development phase which has become pathologically "eccentric" and conditioned as a coping mechanism?

It has indeed been a rare event in psychology and psychiatry when clinical problems which have been considered rather different are brought in relation to each other. For this reason we give the following from Scheerer, Rothmann, and Goldstein (**52**, p. 57):

Recently Kanner has discussed 11 cases of personality deviation in children in terms of "autistic disturbances of affective contact" which he considers a special syndrome. Pointing to the misplaced eagerness of parents to promote precociousness in their children he states: "Their excellent rote memory, coupled with the inability to use language in any other way, often led the parents to stuff them with more and more verses, zoologic and botanic names, titles and composers of Victrola record pieces, and the like. Thus, from the start, language, which the children did not use for the purpose of communication—was deflected in a considerable measure to a self-sufficient, semantically and conversationally valueless or grossly distorted memory exercise. To a child 2 or 3 years old, all these words, numbers, and poems (questions and answers of the Presbyterian Catechism; Mendelssohn's violin concerto; the twenty-third Psalm; a French lullaby; and encyclopedia index page) could hardly have more meaning than sets of nonsense syllables to adults. It is difficult to know for certain whether the stuffing as such has contributed essentially to the course of the psychopathologic condition. But it is also difficult to imagine that it did not cut deeply into the development of language as a tool for receiving and imparting meaningful messages."

Kanner's behavioral observations in these children represent new valuable material for mental pathology. Since his case histories show many parallels to those here presented it may be in order to make some comparisons between his interpretation and our hypothesis on the role of concreteness in defective children and their retentivity.

According to Kanner "the outstanding, pathognomic," fundamental disorder is the children's *inability to relate themselves* in the ordinary way to people and situations from the beginning of life" (p. 242). He explains all behavioral abnormalities found in these children from their *affective* disturbance, from their "desire for aloneness and sameness." To Kanner the inconsistent picture of intellectual ability, the obsessive repetitiousness, the shock reactions to loud noises and moving objects, and the "truly phenomenal memory" in these children is accounted for by their emotional resistance against change in the outer situation—the insistence upon "identical spatial or chronological order." In following Kanner's impressive observations in support of his view, it appears nevertheless as if Kanner has neglected the qualitative nature of the intellectual abnormalities in this picture. The

case histories abound with instances of compulsive concreteness in thought and action. In our opinion this is only explicable on the basis of an impairment of abstract attitude which is intimately bound up with the affective disturbance. To mention only a few problems, it is hard to see how an affective disturbance alone can account for what Kanner calls the "literalness" symbol of affirmation, detached from the specific situation in which it had been acquired; their inability to understand prepositions in the abstract sense. (Asked to put something down, the child puts it on the floor—understanding the word only in the originally acquired situational sense.) It is hard to follow Kanner when he makes the affect-anomaly responsible for: "the absence of spontaneous sentence formation and the echolalia type of reproduction, which in every one of the eight speaking children has given rise to a peculiar grammatic phenomenon. *Personal pronouns are repeated just as heard.* The child once told by his mother "now I will give you your milk" expresses the desire for milk in exactly the same words. Consequently he comes to speak of himself always as you and of the person addressed as I." We have encountered this reversal of pronouns in three cases here presented all of which showed pronounced impairment of abstraction.

This peculiar "grammatic" phenomenon appears to be more than a mere grammatic one or a purely mechanical echolalia. The child hears himself addressed as "you" and the other person speaking of himself as "I." Only on a concrete level of thinking is the literal application of the word "you" to the child himself and "I" to the other person explicable, because the child cannot detach the words from their experienced "belongingness" in the actual situation and reverse this belonging in terms of a relational symbol. (The corresponding phenomenon in normal children is their frequent use of their first name or the third person in referring to themselves.)

Is the child's inability to shift the word "you" from himself to the other person, and the word "I" from the other person to himself, is this inability to grasp the relational meaning of "you and "I" in the abstract, merely the result of the affective disturbance or not a symptom of impaired abstraction and limitation to the concrete as well?

In discussing their peculiar memory Kanner speaks of parrot-like repetitions of heard word combinations, of "delayed echolalia."

As in the case of L., "the children had learned at an early age to repeat an inordinate number of nursery rhymes, prayers, lists of animals, the roster of presidents, the alphabet forward and backward, foreign (French) language lullabies ... even long and unusual words were retained with remarkable facility." Yet in contrast to these recitals, their spontaneous language-development and understanding was retarded. In Kanner's concepts it is the need for "sameness and autistic aloneness" that sufficiently accounts for both, this semantic retardation and for the abnormal retention of verbal material, which latter he characterizes as completely senseless for the children. This makes it quite difficult to understand *why* they so eagerly and readily absorbed and reproduced such material, and even liked to spell out words. Is perhaps the fact that the children did not grasp the meaning of language in the normal way, the motive for their heightened responsiveness to and their tenaciously obsessive reproduction of phonetic sound patterns? In the light of our own case-material it seems highly probable that these children excelled precociously in verbal memory for the same reasons as we outlined in our hypothesis. And the question may arise, whether the disturbance in affective human contact they suffered is not secondary to the defect in abstraction or parallel to it. Perhaps this hindered a normal grasp of the semantic aspect of language and impelled these children to cling to that aspect of speech which was concretely sensible and apprehensible for them in terms of auditory motor patterns (52, p. 57).

The similarities between the autistic child and the idiot savant are indeed striking. We feel, however, that there are several questions which seem to pose difficulties for an explanation based primarily, if not exclusively, on an impairment of abstraction.

How is such an explanation to explain (or to be integrated with) the impression, reported by Tredgold (56) and supported by our own review of the literature, that the large majority of idiot savants are

males? There also seems to be a preponderance of males among the reported cases of infantile autism.

There appears to be no grounds for doubting that there is an impairment of abstraction in both the idiot savant and the autistic child. When Scheerer, Rothmann and Goldstein cautiously suggest that in these cases we may be dealing with a lawful modification of a normal development phase which has become pathologically "eccentric and conditioned as a coping mechanism," at least two questions arise. Is the coping mechanism always a response to the impairment of abstraction—that is, the impairment of abstraction is always the *etiologic* factor "forcing" the child to develop atypically? Is development normal up until that phase when the organism should be able to respond abstractly? To answer either question in the positive would certainly conflict with the experience of those clinicians who report having discerned the characteristics of autism well before the period when abstraction as an intellectual characteristic normally becomes manifest (41, 50). In other words, factors other than intellectual seem, in some cases at least, to be primary.

To explain the idiot savant and the autistic child primarily in terms of an impairment in abstraction would be more plausible if these were cases with demonstrable central nervous system pathology. In the published literature many, if not the large majority, of these cases have no such discernible pathology. Because these conditions can and do appear in the presence of positive neurological evidence of brain injury does not allow one to assume that where such evidence is lacking it is because of the crudity of neurologic procedures. This may be the case but the burden of proof would seem to be on those who make the assumption. Because schizophrenia can follow alterations in the central nervous system does not mean that all schizophrenia can be explained in this way.

Perhaps our most serious reservation to Scheerer, Rothmann, and Goldstein's discussion of the idiot savant and the autistic child concerns their failure to consider the possible ways in which familial personalities and organization either interact with or cause or exacerbate the impairment of abstraction. For example, Kanner, who has reported the largest series of cases of infantile autism, comes to the conclusion that the personality of the parents and their techniques of child rearing are not irrelevant factors in attempting to understand the autistic child. It may be that Kanner's sample is in some ways a biased one but until this is demonstrated one cannot dismiss the possibility that factors external to the autistic child are important in the development of the condition. In this connection it might be pointed out that in the case of the idiot savant which Scheerer, Rothmann, and Goldstein describe there is practically no discussion of the possible role of parental personality on the child. We point this out because of our impression that what little is contained in the monograph on parental personality and behavior suggests similarities to Kanner's descriptions of the parents of his cases. Unfortunately (but not unexpectedly) in the entire literature of the idiot savant there is not a single acceptable personality description of the parents. We are here not contending that parental personality and behavior is the etiologic factor; for reasons to be discussed later we feel such a statement to be unjustified on theoretic grounds. However, for similar reasons, we are contending that any explanation of behavior which is based exclusively on factors "inside" the individual is likely to be a very incomplete one.[10]

[10] In their discussion of very young autistic children, Ritvo and Provence (50) state: "We do not in our thinking neglect the part the mother's attitude plays in this condition. We believe that the child's disturbance sets in motion a circular process with child affecting mother and mother in turn affecting child. We would like to conclude

If one were to assume that the kinds of cases we have been discussing were in some way primarily a function of non-environmental factors (*e.g.*, brain-injury, genetic, constitutional, etc.) then one would expect that they would occur in any culture, *e.g.*, the so-called primitive ones. There is no evidence that this is the case. However, since anthropologists have not been interested in these types of problems, and are not trained to pick them out, the absence of evidence in this instance means little or nothing. We would suggest that cross-cultural studies of infantile autism and idiot savants might be fruitful regardless of the direction of the evidence which would emerge.

In summary, Scheerer, Rothmann, and Goldstein have made an important contribution in pointing out and discussing the similarities between the autistic child and the idiot savant. There is little doubt that in both these conditions there is an impairment of abstraction. In raising questions about the adequacy of such an impairment for explaining these conditions, we have tried to suggest that such an explanation does not seem to cover what apparently are certain facts, particularly the sex differences in the incidence of the two conditions. In addition, we expressed the opinion that discovery of those factors which antedate and are related to the impairment of abstraction may be of vast importance of our understanding of the early development of inteliectual functions—their nature, course, and relation to the external environment. The importance of these cases to the development of a science of psychology would seem to be vastly beyond what their relatively rare occurrence in the general population would suggest.

We hope it is clear from the previous discussion that we are dealing with conditions the etiology of which is very ambiguous. These cases are certainly not homogeneous in terms of presence and degree of organic pathology. It is also impossible to do other than grossly speculate about the possible role of environmental factors. It seems fair to say, then, that neither in the case of infantile autism nor in the idiot savant can one justifiably ascribe the condition to either environmental or organic pathological factors. It would indeed be surprising if the ultimate explanation required knowledge of but one of these factors—the conditions seem far too complex to hold out hope for such simple explanations.

There is one variable, however, which seems not to have been given the attention in theory and research which it seems to deserve. We might begin our discussion of this variable by posing a question we raised earlier in relation to Bourne's "protophrenia" (see page 88): Why is it that some children who have been subjected to the kinds of experiences and relationships which Bourne describes do *not* develop the picture of severe subnormality he calls protophrenia? It probably would not be difficult to find children who have been subjected to even worse experiences and relationships but who do not manifest such an extreme picture. Assuming that there is a relationship in Bourne's cases between certain environmental factors and extreme deficiency, we are unable to explain why more children do not develop this condition. We are faced with the same question of selectivity of occurrence with the idiot savant and infantile autism, regardless of whether one assigns primary importance to organic or environmental factors.

While we would agree that the organic and environmental factors have to be viewed in relation to each other, the variable we have in mind is perhaps independent of both, at least in the earliest months of life. We are here referring to something akin to what Bergman and Escalona (5) have called "unusual sensitivities in very young children":

> It was several years ago that the authors were first struck by the observations to be reported

with a remark made by the mother of one of these children when told of her child's need for continued stimulation from her: 'Why should I pick him up when he doesn't even smile at me?' "

here. Some very young children possessed unusual sensitivities manifesting themselves in several, if not in all, sensory modalities (visual, auditory, tactile, etc.). Colors, bright lights, noises, unusual sounds, qualities of material, experiences of equilibrium, of taste, of smell, of temperature, seemed to have an extraordinarily intensive impact upon these children at a very early age. They were "sensitive" in both meanings of the word: easily hurt, and easily stimulated to enjoyment. Variations in sensory impression that made no difference to the average child made a great deal of difference to these children. They were also characterized by a certain precocity, though this was very unevenly distributed among the diverse functions of their personality. The first impression which some of their reactions and abilities gave was that of unusual giftedness such as might be observed in the budding of a genius. Further observation, however, suggested comparison with individuals suffering from a traumatic neurosis, or a psychosis, and even with feebleminded children. Closer study and follow-up then made it appear that childhood psychosis was the fate of these children, though we are not sure yet that all children of the type to be described eventually develop a clear psychotic picture . . .

If we examine more closely those facts that impressed us as bespeaking unusual sensitivity in the described children, we become aware that they differ from each other in several respects and can be grouped accordingly. We find that we have observed facts pertaining to many parts of the sensorium, *i.e.*, to visual, to auditive, to tactile, to olfactory, to gustatory, to equilibrium, and to temperature experiences. Some of the children reported on reacted very sensitively to light or colors, to noises and music, to materials that came in contact with their skin, to smells and perfumes, to foods, to rocking and swinging, to to cold air or cool objects. Thus, one obvious principle of grouping our observations is furnished by the sensory modality.

Then we find that what impressed us in some observations was the reaction to the intensity or quantity of stimulation, while in other cases the observation is more easily understood as a reaction to quality. Thus if any kind of slight sound seems to awaken a sleeping infant, or arouses a reaction from the waking one, we will consider that he reacts to the intensity of the stimulation, in fact here to a very low intensity. But if certain sounds or combinations of sounds attract a child, while other sounds or combinations of sounds of equal loudness repel him, it seems plausible to consider this a reaction to quality. Other reactions to quantity that we find in our material, are, *e.g.*, reactions to light of a certain brightness, reactions to normally imperceptible (or at least not usually reacted to) amount of odor, reactions to slight disturbances of the equilibrium, to slight impressions on the feeling of temperature. On the other hand, observations pertaining to certain colors, certain materials, specific odors, foods, we can group with reactions to quality. Whether a special fondness of rocking should be grouped with reactions to quality or to quantity may be debatable. With some sensory modalities this distinction does not seem to make much sense. We would not be able to say, *e.g.*, what a reaction to quality would be like in the modality of the sense of temperature.

Bergman and Escalona go on to point out that unusual sensitivities in young children may result in premature defensive reactions which are inadequate in the face of later trauma. "The hypothesis will be offered that the infant who is not sufficiently protected from stimuli either because of a 'thin protective barrier', or because of the failure of maternal protection, may have to resort for such protection to premature formation of an ego. When this premature ego breaks down, possibly as a consequence of a trauma, the psychotic manifestations are thought to set in."

Bergman and Escalona's clinical observations and conclusions allow one to raise several questions: (a) To what extent do autistic children have a history of unusual sensitivities?—a question which Bergman and Escalona raise but which existing studies cannot answer. (b) To what extent would the histories of idiot savants indicate unusual sensitivities preceding the appearance of unusual intellectual behavior? (c) To what extent do the children described by Bourne have unusual sensitivities? *Perhaps of more significance than these questions is the assumption that the crucial factor in*

these types of atypical development is the interaction between unusual sensitivities, on the one hand, and environmental factors, on the other hand. Whether the infant with unusual sensitivities develops atypically may be a function either of parental handling of these sensitivities or fortuitous organic conditions or some combination of both.[11] In other words, not all children with unusual sensitivities would be expected to develop atypically. Similarly, objectively similar environments or organic pathologies would have differing effects on individuals who differed in terms of the unusual sensitivities described by Bergman and Escalona. For example, the "protophrenics" described by Bourne may be those who had unusual sensitivities, while other children with similar or worse experiences and relationships but who did not show such extreme subnormality may not have had such sensitivities.

It is reasonable to assume that sensory hyperreactivity is but one of several ways in which the young child may be unusual. Put in another way: the very young child can be viewed in terms of various behavioral continua, and extreme placement on any of these continua, as in the case of unusual sensitivities, may be the predisposing factor which when interacting with environmental or organic factors has untoward effects. The work of Fries (17–19, 42) on "congenital activity types" supports the contention that the study of temperamental variations in the very young child may provide at the least a partial answer to the question why different children are differentially affected by similar external events or similar organic conditions. This is clearly not a problem of peculiar significance to the area of atypical development but one which is truly basic to our understanding of normal development, the nature and range of the earliest individual differences in relation to rearing and educational techniques.

[11] The occurrence in the same child of an unusual sensitivity and an organic pathology does not, of course, mean that the two are causally related, although this may be the case in certain instances. The presence of an unrelated organic pathology may make it more difficult for the child (and parent) to cope with the sensitivity. For example, Frankl (16) described a case of severe autism in a child with tuberous sclerosis. Although most cases of tuberous sclerosis are severely defective, there are some whose intellectual functioning is less, or not at all, affected by the condition (57, p. 278). In any event, extreme autism is not a marked behavioral characteristic of these cases. The extreme picture of autism which Frankl describes need not be related, in its initial phases at least, to the central nervous system pathology. It is conceivable that their origins are independent but that the central nervous system pathology exacerbates the autistic tendencies.

LIGHTNER WITMER

If only for historical reasons we feel compelled to say something of Witmer's work. Psychologists, psychiatrists, and educators of today may know of Witmer's name in connection with the fact that he organized the first psychological clinic in this country in 1896 at the University of Pennsylvania. What has been forgotten, unfortunately, is that in the early years of this century he was concerned with and wrote most illuminatingly about the differential diagnosis between mental deficiency and childhood psychosis. Years before the autistic and schizophrenic young child was described and recognized by American psychiatrists, Witmer published his "orthogenic" case reports in which the major characteristics of this type of child were delineated. To be sure, Witmer's descriptions are not as sophisticated or complete as those of today, but the serious reader who studies Witmer's writing will probably agree that he must be considered one of the real pioneers of American child psychiatry.[12]

Aside from his awareness of the importance of differentiating between mental defect and childhood psychosis, the signifi-

[12] In 1907 Witmer started the Journal called "Psychological Clinic." It is no longer in existence. Some of his case reports (60, 61, 62, 63, 64, 65) are more concerned with subnormality than others. Throughout the journal will be found similar cases by his students. As would be expected, there is much in the journal which is now outmoded, but the serious student of the problem will gain much by scrutiny of all the volumes.

cance of Witmer's work lies in his therapeutic approach to and success with these instances of differential diagnosis. We give below excerpts from one of his reports (64):

At the age of two years and seven months this boy responded to every test like a feebleminded child and he was diagnosed by competent experts as feebleminded. Today he is a normal boy, not quite seven years old, reading, writing, and doing the number work of the second school year. Either he was not feebleminded and the diagnosis was a mistaken one, or feeblemindedness can be cured. What is feeblemindedness—a performance level or an irremediable mental defect? Don's response to treatment shows that he had grave but not irremediable defects. His arrest of development was nearly complete, the results of disease and the psychosis which accompanied the disease.

When a normal adult becomes insane we observe a marked change of character. "He is no longer himself," we say, and a prominent sympton is a reduction of mental level called technically "dementia." Auto-intoxication, disease and shock may cause insanity. Let us suppose that one or all of these causes affect a child in his first or second year. We shall not be able to observe much change in the child's mentality except that his mental development will be arrested. I maintain that one type of feeblemindedness, better called arrested development, is due to the same causes which produce insanity in an adult, and that in some cases the psychosis or mental disorder can be cured and the child restored to completely normal condition, provided the case be taken in hand early enough.

Except to the very observant eye of an experienced expert, these cases look more like feeblemindedness than insanity. Nevertheless, they are a species of feeblemindedness or insanity, whichever name we choose to apply, very different from the congenital imbecile, one of the mongolian type, for example. The mental disorders of children which cause arrest of development and apparent feeblemindedness are as diverse as the mental disorders observed and classified by the alienists. A child may be either feebleminded or insane, or he may be both feebleminded and insane. Some of the Orthogenic Cases reported in the earlier numbers of the *Psychological Clinic*, notably Orthogenic cases Nos. 4, 6, 12, and 13, are not primarily cases of congenital defect, but cases of mental disorder in which there is a greater presumption of possible cure than in the cases of the child who is both qualitatively and quantitatively feebleminded.

... He was five years old last July, and so I entered him the following autumn in a near-by school, where he is the youngest of a group of first-grade children. His teacher says that he reads better than any of them and, except that he is poor in handwork, she considers him as competent as the other children.

"Terrence," said he to his pal, the gardener, who was taking him to school the first day, "don't call me Donnie when we get near the school; don't call me Donnie or Don; call me Donald, which is right."

I saw Donald for the first time when he was two years and seven months old. His father carried him into my office, and deposited him, a soulless lump, upon the couch. He sat there with the stolidity of a Buddhist image, absorbed in the inspection of a card which he held in his pudgy hands, as regardless of his father and mother as of the new objects about him. While his gaze moved over the card, he scratched the back of it gently and incessantly with his finger nails. At times he gritted his teeth; and then again he made a crooning, humming sound with which it is his habit to lull himself to sleep.

He paid no attention to a rattle, to a bright-colored ball or to a picture book which I held before him, but every effort to remove the card from his hands he resisted. His face, already crimson, became empurpled. His physiognomy took on an expression of angry hostility; and I retreated before the approaching storm, leaving him again to his absorption in the card.

"He is fond of music," his mother said; but the liveliest strains of the talking machine were powerless to distract him from his chosen preoccupation. In the months to come I was to discover that by preference he would sit or lie in bed for hours, looking attentively at the object which he happened to be holding in his hands. It appeared to be persistent, concentrated attention, that most difficult and valuable of mental powers to cultivate.

From two to six years the child has the flitting attention of a monkey. "How do you select your

monkeys for training?" a trainer of animals was once asked.

"I hold a lighted match before them," he replied, "and picking out as the easiest to train those that look longest at the burning match."

Donald would look at nothing but his card. One could not guess what lay behind those dull blue eyes. Was it interest, or only emptiness of mind—the dreamy listlessness with which the corner loafer looks at the passing world?

"What are those abrasions about the mouth and ears?" I asked.

"When he gets angry," his mother said, "he will scratch and tear at them."

"What else can he do?" I asked, not venturing to break in upon this obstinate immobility by trying to get him to perform the simple task which might, perchance, reveal some hidden mental ability.

"Can he walk?"

"A little, but he only began about two months ago," she replied. "Until he was over two years old he hadn't even crawled; and he only learned to crawl by his nurse taking hold of his knees and advancing them one after the other."

As the flower blooms, the fish swims or the bird flies, so the child crawls, walks and talks. It is the unfolding of his own instinctive impulses. But this child had to be taught to crawl and to walk, and even yet he could only toddle about uncertainly. If he fell upon his face he would lie helplessly crying with his nose to the floor. Either he did not have the strength to change his position, or he did not know how, or he was unwilling to make the effort.

He never uttered a word spontaneously, and he could repeat at command only a few words like "Kitty," "Mamma"—eight words in all. His understanding of language seemed to be limited to pointing to his head, eyes, ears and nose when these words were spoken. Even a chimpanzee of the same age as this boy, if brought up in human surroundings, will give evidence of understanding more of spoken language than this boy did. He could not feed himself. A much younger child can hold a cup or a spoon, but this boy could not even close his lips upon a cup when it was offered to him. He was still in diapers, and weeks were to pass before he could be safely clothed like the normal boy of two years and a half.

At two years and seven months Donald was doing no more than many a child does at twelve months, no more than every child should do at fifteen months. No one who saw him needed to consult an expert before deciding that he was subnormal. You had only to look at the large head—"top-heavy Bill" one of his teachers called him—the fat red face, the expressionless eyes and the helpless body, to arrive instantly at the conviction that "this child is feeble-minded."

And feeble-minded I thought him—of such low grade that I refused at first to accept him for educational treatment in my school. With reluctance I finally yielded to the parents' pleas. He was the youngest child I had ever accepted for psychologic treatment, and apparently the most hopeless.

The expert, like the parent, bases his opinion on the child's appearance, behavior and history. But even more important than these is the "*attempt to teach.*" In doubtful cases I do not like to express an opinion until after I have observed the results of attempting to teach the child something new. This can often be done at the first examination, but I could not even begin to teach Donald.

"I should like to see him walk," I said. But when he was lifted from the couch, put upon his feet and made to walk, he burst into a paroxysm of rage. His eyes became bloodshot; even his gums bled. When he was put back upon the couch he returned to his contemplative absorption in the card. Offered a block, he made no effort to take it. He even closed his eyes, as though the very sight of it and me were more than he could endure.

When I took the card away, so as to secure his undivided attention, he had another paroxysm of rage. From this, however, I derived a little hope, for passion and rage may be an expression of strength. The child at least had energy at his disposal. His violent resistance evidenced resolute determination. Obstinate children are better material for training than the overpliant sort. I looked at him, sitting impassive but always bold upright, and this too, I thought an encouraging sign.

"He is a very easy child to neglect," one of my teachers entered in her report soon after he came to the school. "If you let him alone he will sit or lie in bed for hours and give no trouble. It is only when you try to do something with him, to dress him, or bathe him, even at times too feed him, that the trouble begins."

It takes some time and care to adjust a child to new surroundings, so I considered it no great misfortune that Donnie promptly got the measles. For a couple of weeks it was necessary to isolate him in the care of a trained nurse. This probably helped to make him less resistant to strangers. Perhaps there also awoke within his soul some responsive feeling of gratification when the soothing hand of the nurse or the doctor brought him relief from his distress of body. One month after Donnie's arrival I began his education.

"What to do," and "How to do it" are two puzzling questions confronting teacher and parent at every turn. To answer the first question is to present the aims of education. In the early years of education the three R's are the chief objective. The answer to the second question, "How to do it," will determine our method of procedure.

Educational aims and practice are commonly the outcome of theory. For example, an interesting and important theory of recent origin is the Montessori method. It aims to develop a child's natural abilities. It also has a theory of educational practice. It emphasizes and, in the opinion of many, relies exclusively upon appealing to the child's natural inclinations and desires. Deprecating the use of constraint and force, it throws the reins over the neck of the horse. Several children have been brought to me for examination and educational treatment who were nearly ruined by too close adherence to this supposed Montessori method.

I hold that constraint and liberty have equal value. At one time constraint, at another liberty, will bring the best results. The wise employment of constraint and force calls for greater intelligence and judgment on the part of teacher and parent than the leaving of the child free to work out his own salvation and development.

I try to approach the problem of educating a child like Donald without any preferred theory. More than twenty years of experience has led me to see that there is some good in most theories. A few are fit only for the scrap heap. One guiding principle, however, has stood the test of time and use: "The first task of teacher and parent is to gain and hold the child's attention by giving him something he *can* do, and after that, something he *can't* do": this in general is my method.

My educational aim is to develop attention by choosing tasks which develop it. Whether a child be one year of age, or two years, or six, whether he be in high school or college, the guiding principle of the educator should be to gain and hold attention first, and then to cultivate concentration, alertness, persistence, and endurance, all of these being attributes of attention.

For the rest, I feel my way. I watch the child to discover what he does with interest and with ease, and from here I get him to take a step forward in the direction best calculated to bring him to what I am aiming at, "the next higher level of attention." Montessori provides the child with stimulating objects—here didactic material—and leaves it with the child to make the next step forward. This is doubtless an acceptable procedure; but suppose the child refuses to take a step in any direction. He must be shoved.

To shove a child in the direction you want him to go is easy if the child is pliant and submissive. If he is a fighter like Donnie, and if, like him, he has no desires except to be let alone, the development of attention and the enforcement of obedience must go hand in hand.

When you have a trout on a hook at the end of a thin line, the only way to land him is to play him. He is lively and vigorous. He has desires which conflict with yours. If you use too much force you will break the line. If you use skill, yielding and yet constraining, you will in time get him into your basket. In this way the skillful teacher "plays" the child. The hook of attention is attached to the line of obedience, and then she watches the child's every move to insure his advance in the required direction. Shall she coax or force him? On the lee shore of this question many a gallant education craft lies shipwrecked.

You can coax most children, some of the time at least, by appealing to their interests and desires, even as the hunter entices the deer to come within gunshot by appealing to its curiosity. But some children can't be coaxed, any more than you can wheedle a trout into your basket.

For example, take Donald. He did not have a keen desire even for food. He would not eat prunes, apparently because he disliked their appearance, and so they had to be mixed with his cereal in order to get him to eat them. He would not drink milk or water from a transparent glass. It must be offered to him in a cup. In the early days, indeed, he declined to drink water at all, and got his only liquid in the shape of milk or soup.

He declined to accept a sugarplum offered as a reward of merit; and if you took away the object he so fondly clasped in his hands, and then yielded to his ragings and returned it to him, he would very likely throw it violently across the room. He disliked to be dressed. He disliked to be taken out of bed and put on the floor. He disliked to be taken for a walk.

All these things aroused angry resistance; and in his passion he went so far as to do himself bodily injury; but as long as Donald held something in his hand there was peace and quiet.

"What to do with him?" He could not be bathed and dressed in this happy state of calm contemplation. Take away what he held and his hands went up to his ears and mouth, tearing at them till they bled. Tell him to keep his hands down, they went up just the same; perhaps he only scratched himself a little more strenuously. Put mittens on him, as his former nurse did, and he still went through the motions.

Smack his hands, anger and passion intensified the violence of his resistance. The only thing to do was to hold his hands. Could he be compelled to keep them down after they were released? The historic battle lasted for an hour and a half. His hands were held while his teacher spoke to him from time to time: "If I let your hands go, will you keep them down?"

He raged, he stormed, he grew apoplectic, but the hands were firmly held. At every lull in the storm they were released, and up they went again. In the end he gave in. Ninety minutes showed remarkable endurance, determination and consistency of purpose, qualities which might be successfully employed in his educational development later.

Never again did Donnie hold out for so long on this or any other issue. My records show that though he raged at intervals during the ensuing twelve months, the longest period of resistance lasted for ten minutes only. He had learned his lesson. There was an inevitable persistance that would outlast his own. He might as well give in first as last.

The subsequent development of this boy under Witmer's supervision was both dramatic and heartening. That Witmer was an astute observer who did not allow dramatic change in the patient to cause him to overlook problems and weaknesses may be gleaned from Witmer's concluding remarks:

If I began my work without a theory and without understanding of Donnie's mental status, I am far from that position now. I have unraveled much of the mystery, and I find the understanding of this one child of important value in interpreting the behavior and progress of other normal children. I believe that Donnie was at the start dominated by fear, which plays still an important role in his behavior. His concentration on the card was in the nature of a defensive reaction. He disliked to get out of bed because he was afraid to get out of bed. He disliked to walk and talk because he was afraid, perhaps of failure.

It was noted on one occasion that when taken outdoors he would not stop screaming even after he had been put on the back of a pony. I know now that this was the worst thing that could have been done to him. Donnie is afraid of all animals. He takes kindly, however, to little creatures and has often alarmed his teachers by bringing them caterpillars and worms.

One day Donnie, while seated at a table playing with a train of cars, had his attention called to the fact that a little gray kitten was in the room. He was mortally afraid of it, so he would not turn his head to look, but kept moving the train back and forth, saying "Puff! Puff!" in the same absorbed concentration which was characteristic of him at the beginning. He was ignoring the kitten just as he used to ignore people he disliked by closing his eyes when they came into the room.

He was afraid to look down a well, he was afraid of a doll, of a soft rubber ball, of a balloon, a loaf of bread, a spinning top. He was afraid to go on a sail-boat the first time, but the second time he went with joy. He took a fearful pleasure in trains, for he loved them as moving things, and yet they terrified him. He would say: "Let us go to town in the three trolleys"; but when you asked him why he would rather go in the trolleys than in a train he would never tell you.

He has never verbally admitted that he is afraid of anything. "Won't hurt you," he very early exclaimed whenever he was frightened by anything; and this was one of his first spontaneous reflections. "Don't have to pat the pony," he would reiterate during the many weeks required to get him to overcome his fear of the school pet.

The effort to take him out driving in a little pony cart, which it was thought would entertain him, only succeeded after a period of two months. But then, as was usual with him, he couldn't get enough of driving behind the pony.

Even yet he is afraid. "I like dogs," he declared lately, as he started on his way to school. "Nice kind dogs which don't bite," he added thoughtfully. Nevertheless, he managed unobtrusively to place his companion between himself and every dog. "I like to pat dogs," he boasted; but when one appeared unexpectedly he excused himself tactfully: "I don't like them that color."

So, while Donnie is fearful, he is not a coward. He is doing his best to overcome his fears, and he has worked out his own method of doing this. He had no fear of dark or of the supernatural.

Fears and desires are the two greatest motive forces of mankind. No problem is more perplexing and none so absolutely fundamental as the proper treatment of fears and desires so that these motive forces may excite the actions desired. As I understand Donnie now, he had no desires, but many fears. We compelled him to do those things which he feared. As soon as he had done the fearful thing, the fear, in many instances, disappeared and desire took its place. Donnie is now afraid chiefly of what surprises him.

Donnie's obstinacy measured the intensity of his fear, but in part it measured also the intensity of his desires. Always, from the very beginning, Donnie has known just what he wanted. Never was there any wabbly uncertainty of choice. He either desired it or he didn't desire it. This, to my mind, is a strong and valuable trait of character if you can turn it to the right use.

The desire for possession gives rise perhaps to his keenest pleasure. He held on to his card, not only because it enabled him to ignore the fearful things of the world about him, but he held on to it because here was something "all his own." Not until recently has he been willing to share any of his possessions with others. For a long time he not only clung passionately to his own possessions, but appropriated the playthings of all the other children as well, so much so that his room was known as the "Robber's Den." He is now so far advanced on the road to generosity that he will give away his second-best toy.

He has always shown the same concentration of attention which he showed at the beginning. One day recently he wore to school a necktie which he had borrowed from the gardener, Terence. The teacher could do nothing with him that day because he persistently explored the attributes of his new possession. He met Terence, who came to take him home, with the matured fruit of his morning's work: "Terry, can you see the top of *your* necktie?"

His first craze was for automobiles, and then for sailboats, bicycles, trains and cars—anything that moved. As he learned to talk, he went through the magazines. "It's an automobile, see the automobile," he kept reiterating. When he grew fond of excursions abroad, "Are we going out, Agnes?" he would say, "Agnes, are we going out?" a thousand times until he threatened to drive his nurse to distraction. No child can have a better endowment for future accomplishment later than this power of persistent concentration.

Donnie's traits of character are therefore positive traits. He has a definite array of abilities, keen desires, self-dependence. Even from the first he preferred to walk alone, though in constant fear of falling, rather than hold someone's hand. He only sought the hand if a terrifying object came in view. With strong desires and fears, strong likes and dislikes, Donnie has an equal capacity for happiness and great unhappiness, for success and failure. He can be sweet-tempered or angry and resentful. His emotional balance is easily disturbed, and he still requires very careful handling.

Of the cause of Donnie's mental condition when he came to us, and which led several experts to diagnose him as feebleminded, I cannot be sure. He had an illness after birth, which I now believe left his brain so devitalized that it permitted fear to gain the upper hand over desire. Of one thing I am certain: If Donnie had not been given the painstaking and expert training to which we subjected him he would by now have fallen into a state of irremediable feebleminedness.

Although today the term psychotherapy refers to a variety of techniques and interpersonal relationships, it is most likely that no "school of psychotherapy" would advocate Witmer's initial handling of this boy. It would be quite wrong, obviously, to view his initial approach as a punitive one (although from the standpoint of the boy this

was the case) in the sense that it reflected Witmer's feelings and did not stem from any theoretic conceptions. By focussing on the boy's inability or unwillingness to respond to an external figure, Witmer was deliberately attacking in a direct and dramatic manner the most blatant symptoms, *i.e.*, the autistic-like way of relating to the external world. Equally important, we think, is that this child was placed in what was essentially a therapeutic-educational milieu in which all phases of his behavior were under scrutiny, supervision and stimulation. One gets the impression of a total push situation which went far beyond what today takes place in individual psychotherapy with these kinds of cases (on those relatively rare occasions when such treatment is attempted). The fact that the above case is but one among many reported by Witmer and his students would seem to have implications for current attitudes toward the treatment (or, more frequently, the non-treatment) of the very young, withdrawn, autistic-like and defective-like child.

There are two things which impress one in Witmer's work. First, Witmer seemed to employ therapeutic effort as a way of deciding as between one or another diagnostic possibility—most frequently between normal or defective potential. Second, the fact that the child with whom he was working was subnormal, and in some cases apparently severely defective, did not seem to influence the intensity of Witmer's therapeutic efforts. One gets the impression from his writings that Witmer, like Itard, was not discouraged by outward appearances and was possessed by a fierce determination to demonstrate that in the most hopeless case there was unrealized potential which could be made manifest under proper guidance. Such an attitude may be a mixed blessing and result in failure and harm to others. But when in the case of people like Itard and Witmer such an attitude is combined with brilliance and creativity—and a remarkably painstaking attention to detail and method—one gets the kinds of results that shed real light on human potentialities. Research reports like the present one are far less needed in the area of subnormal functioning than people like Itard and Witmer. Freud did not need somebody else's research report to make his now recognized contributions to psychology and psychiatry and, in their own fields of interest, neither did Itard and Witmer.

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ON THE PARTICIPATION OF ADAPTIVE HORMONES IN DISEASE SUSCEPTIBILITY

Illustrated by the Acquisition of Resistance to an Experimental Nervous Disease during Adaptation to Cold[1,2]

HANS SELYE, M.D., PhD.[3]

Ever since the description, in 1936, of the General Adaptation Syndrome (the body's response to nonspecific stress), it has been the principal aim of investigators interested in this field to clarify the mechanism through which, depending upon circumstances, stress can either produce, or prevent, disease. During the last twenty years, it has been amply demonstrated that in response to a variety of stressors (trauma, infections, intoxications, nervous stimuli, etc.), the body reacts in a rather stereotyped manner, by the secretion of certain hormones such as ACTH and corticoids. The resulting excess in these "adaptive hormones" is an essentially useful defense reaction. However, under certain circumstances, it can become the cause of disease or, at least, predispose the body to the production of morbid changes. For example, the excessive secretion of ACTH and of anti-inflammatory corticoids is useful because it helps the body to survive during an emergency and because it suppresses excessive inflammatory reactions; but, at the same time, it can also be harmful in that it may enhance the spread of infections, or predispose to the production of peptic ulcers.

From the neurologist's point of view, it is of special interest that certain adaptive hormones can produce severe derangements in the function of the central nervous system. Thus, it is now well known that excessive amounts of ACTH and of anti-inflammatory corticoids can cause euphoria, insomnia, depression and even schizophrenia-like manifestations. Furthermore, an excess of certain corticoids such as aldosterone and desoxycorticosterone may produce a syndrome of hypokalemic paralysis, which greatly resembles the familial periodic paralysis of man. In this connection, it is also noteworthy that, with corticoids and similar steroids, we are able to produce complete surgical anesthesis in animals, an observation which eventually led to the use of steroid anesthesia in human surgery.

All these observations have made it clear that there are close connections between stress and the nervous system: on the one hand, emotional stimuli are eminently capable of stimulating the production of adaptive hormones; on the other, adaptive hormones exert a pronounced influence upon the function of nervous tissue.

The voluminous literature which deals with all these topics has been reviewed in a series of monographs (4, 9), so we need not discuss it here in detail.

One of the most interesting facts that have been brought to light in the course of research on stress is the phenomenon of "crossed resistance," a condition in which exposure to one agent induces resistance to another (3). Investigations have shown that, in most instances, this phenomenon is due to the fact that the stress of exposure to a noxious agent results in a discharge of anti-inflammatory corticoids which, in turn, inhibit responsiveness to other agents. It has been demonstrated, for

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[3] From the Institut de Médecine et de Chirurgie expérimentales, Université de Montréal, Montreal, Canada.

example, that it is through this mechanism that stressors (cold, muscular exercise, trauma or infection) can inhibit the lung edema normally produced by adrenaline, the anaphylactoid reaction usually elicited by egg-white or dextran, and many other types of inflammatory responses (4).

Since the thyroid also participates in certain systemic adaptive reactions, it seemed of interest to determine whether the increased endogenous secretion of thyroid hormones could likewise produce a phenomenon of "crossed resistance." Some time ago we showed that thyroxin injections can prevent the skeletal lathyrism normally produced by certain aminonitriles (*e.g.*, aminoacetonitrile) in the rat (5). This raised the question whether osteolathyrism could also be suppressed by an endogenous excess of thyroid hormone, such as occurs during adaptation to low temperatures. Experiments designed to test this hypothesis have shown that exposure to cold does, in fact, render the skeletal system of the rat highly resistant to the toxic effect of osteolathyrogenic aminonitriles (8).

There are other aminonitriles, for example, $\beta\beta'$-iminodipropionitrile (IDPN), which produce a peculiar nervous derangement. Thuillier and his associates (10) were the first to observe that, in mice, a single injection of IDPN can elicit a permanent derangement of motor activity, characterized by "waltzing behavior," that is, a tendency to run around in circles. In the rat, intoxication with this same compound produces rather similar changes. The animals usually huddle together and appear to be quite normal, but as soon as they are disturbed—even if they are touched only lightly—they jump up and run about in circles as though frightened and disoriented. Sometimes they suddenly jump into the air and then tend to fall out of their cages. Since, during their fall the animals cannot right themselves normally, they frequently sustain injuries upon hitting the floor; this is hardly ever the case when a normal rat falls from a similar height. It is also characteristic of IDPN-intoxicated rats that they make jerky, choreiform movements: upward and backwards, as in opisthotonus, or sideways, as though they were sniffing about to explore something with their bristles. This particular behavior could be due, at least in part, to disorientation resulting from impeded vision, since IDPN also produces severe morphologic changes in the eyes (clouding of the lens, edema, and eventually, detachment of the retina with hemorrhages into the aqueous humor) (5).

It is also typical of IDPN-treated rats that when placed into water they do not swim; they merely turn around in circles and drown themselves, unless they are rapidly removed from the water. Normal rats invariably swim with well-coordinated movements. This "swim test" is being used routinely in our laboratory for the detection of incipient IDPN intoxication. Since the syndrome produced with this drug is primarily characterized by excitement, choreiform movements and "circling," it is referred to as the "ECC-syndrome" (5).

Our interest in this syndrome has been greatly stimulated by the suggestion of some investigators, who felt that IDPN is a "psychopathogenic compound" (2), and that animals in which an ECC-syndrome is induced by IDPN could act as suitable test objects for the assay of tranquilizers and of drugs that might combat parkinsonism and similar motor disturbances. The former compounds could be expected to combat the excitation; the latter, the choreiform movements characteristic of the ECC-syndrome (1, 11). The test is hardly specific, however, because we were able to show that both the ECC-syndrome (7) and the ocular changes that occur after severe IDPN intoxication (6) can be prevented by suitable pretreatment with thyroxin.

In view of these observations, it seemed of interest to determine whether the ECC-syndrome could also be prevented by an excess

of endogenously secreted thyroid hormone, such as occurs during adaptation to cold.

MATERIALS AND TECHNIQUES

Thirty female Sprague-Dawley rats, with a mean initial body weight of 102 gm. (range 92 to 105 gm.), were subdivided into 3 equal groups. Group I was kept at room temperature throughout the experiment. Group II was kept in a temperature of 0°C. during IDPN treatment, and Group III was kept in a temperature of 0°C. for 10 days before and during IDPN treatment.

IDPN ($\beta\beta'$-iminodipropionitrile, also known as aminodipropionitrile, or *bis*-[β-cyanoethyl]-amine) was administered to all three groups, subcutaneously, at the daily dose level of 20 mg. in 0.2 ml. of water during 10 days.

For the objective appraisal of the "ECC-syndrome," we used the "swim test," which was performed as previously described (5). When dropped into a large bucket of water, rats normally swim with coordinated movements; but as soon as the "ECC-syndrome" develops, they circle about in the water aimlessly. This test was performed every 2 days after the initation of IDPN treatment, as well as during the 10 days that followed the discontinuation of IDPN injections.

RESULTS

During the first days, the "swim test" was negative in the animals of all three groups. However, on the eighth day, 40 per cent of the rats in Group I (controls kept at room temperature) gave a positive "swim test", while in Group II (exposed to cold during the experiment only) and in Group III (exposed to cold before and during the experiment), the "swim test" was uniformly negative. On the tenth day, positive swim tests were registered in 90 per cent of the rats in Group I, 50 per cent in Group II and none in Group III. Although at that time treatment with IDPN was stopped, the incidence of positive "swim tests" continued to rise during the subsequent 5 days, at the end of which time, positive "swim tests" were registered in all of the rats in Group I, 60 per cent in Group II, still none in Group III.

During subsequent observations of these animals, we noted no further change in the "swim test" readings. It is well known that the nervous lesions produced by IDPN are permanent, under normal conditions; in our experiments, even continued exposure to cold failed to reverse them.

However, it is evident from these observations that exposure to cold (particularly if initiated prior to the commencement of IDPN treatment) is highly effective in rendering the central nervous system of the rat resistant to the usual toxic actions of this aminonitrile. In order to ascertain that, under our experimental conditions, exposure to cold did, in fact, stimulate the thyroid, the thyroid glands of all 30 experimental animals were fixed in Susa solution immediately after autopsy, and histologically examined on paraffin-embedded sections stained with hematoxylin-eosin. The histologic structure of the thyroids was essentially normal in Group I, while in the two groups exposed to cold, there was marked cellular hypertrophy and hyperplasia.

Since thyroidectomized rats do not withstand exposure to cold, it was impossible to verify the importance of the thyroid gland in the development of this kind of "crossed resistance" by control experiments on thyroidectomized animals. However, we know that small doses of thyroxin can inhibit the "ECC-syndrome," and exposure to cold did, in fact, cause thyroid stimulation under our experimental conditions. Therefore, it appears justified to conclude that the resistance to the neurotoxicity of IDPN that develops during adaptation to cold is probably due to an increased secretion of thyroid hormone.

It is noteworthy that both the bone lesions induced by amino-acetonitrile and the functional derangements of the nervous system caused by IDPN are inhibited by thyroxin as well as by adaptation to cold. Hence, it

is highly probable that the increased secretion of thyroid hormone, in the animals kept at a low temperature, does not selectively protect the bone or the nervous tissue, but probably enhances the detoxification of aminonitriles in the organism.

SUMMARY

These experiments furnish us with still another example of an experimental disease whose development is decisively influenced by a hormone. They show, furthermore, that the amount and type of hormone normally secreted by the thyroid gland during adaptation to cold suffices to induce resistance to a severe experimental disease of the nervous system.

After a brief discussion of earlier work concerning the interrelations between stress, the adaptive hormones and the nervous system, the phenomenon of "crossed resistance" has been subjected to further experimental analysis.

In previous observations, this phenomenon was almost invariably due to the fact that the stress produced by one agent caused an increased secretion of antiphlogistic hormones, and these, in turn, protected the organism against another agent.

The new experiments reported in this communication show that the syndrome characterized by excitement, choreiform movements and "circling" (the "ECC-syndrome"), that can normally be elicited by $\beta\beta'$-iminodipropionitrile (IDPN) in the rat, is prevented by exposure to cold. An analysis of this phenomenon shows with great probability that, there, "crossed resistance" between IDPN and cold is due to the fact that adaptation to a low surrounding temperature stimulates the thyroid, and the resulting excess of thyroid hormone protects against IDNP.

This is yet another instance of an experimental disease whose development is decisively influenced by a hormone secreted for adaptive purposes.

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BOOK REVIEWS

FAIRFIELD, LETITIA. *Epilepsy.* Philosophical Library, Inc., 1957. 159 pp., illus. $4.75

This little volume was written and printed in Great Britain, and was aimed at the general public of that country. Certain sections appear to be directed at physicians as well, especially general practitioners and school physicians. The need for such educational efforts would appear to be the same in Great Britain as in the United States, and it is inferred that many would benefit from a reading of this book. If the Anglo-American similarity is complete, few will trouble to do so.

A considerable portion of the book will be frustrating to the American patient or his family looking for guidance and aid in meeting the tremendous social problems facing the epileptic. Although there is considerable discussion of these problems, this is presented within the framework of the nationalized health services of Great Britain. There is considerable reference to British laws, the educational system, British government agencies, etc. To the British patient, these sections would appear to be of considerable concrete value. To the American, they can only be of academic interest and, perhaps, a source for the generation of envy.

The author points out various shortcomings in the handling of the patient with epilepsy, under the British system. Nevertheless, and without entering into controversy on the merits and demerits of a nationalized health service, this reviewer could only come to the conclusion that, on the basis of the information presented, the epileptic in Great Britain gets a better break than his counterpart in the United States.

There are minor errors of fact and fancy, of typography and bibliographic citation. The book is cheaply printed and bound and its price seems somewhat excessive. However, it will be of genuine interest to the physician and social worker for the picture it gives him of the place the epileptic occupies in the social structure of Great Britain.

J. K. MERLIS

SODDY, KENNETH, Editor. *Mental Health and Infant Development.* Basic Books, Inc., New York, 1956. 2 vol., 597 pp. $9.00

The World Federation for Mental Health sponsored this international seminar on mental health and infant development at Chichester from July 19 to August 10, 1952. The assistant director, Dr. Soddy, directed these seminars which were the first interdisciplinary conferences attracting professional workers from most countries of the world. The first volume includes all of the lectures, and in the second volume thirteen case histories are cited to illustrate the theoretic considerations. The material is based on the studies of children in the first two years of life, and numerous film and reading references are given.

The teaching staff at this meeting included the leading international figures in psychoanalysis, psychiatry, psychology, anthropology, and social welfare: Anna Freud, John Bowlby, Rene Spitz, Margaret Mead, Jenny Aubry, Edith Jacobson, Juliette Favez-Boulonier, Kent Zimmerman, Alan Moncrieff and Helvi Boothe.

The seminars included all phases of infant development, ranging from theoretic interests to definitive clinical entities. The wide range of subjects included formation of object relationships, anaclitic reactions, group identification, effects of technologic change, psychotherapy and comparative analysis of child rearing practices in widely different societies.

In Volume II the editor presents thirteen intensive case histories of children from the United States, Great Britain, and France with different socio-economic backgrounds. If one examines the global picture of the childhood developmental patterns in these

three countries, one is struck by the sameness rather than the differences in growing up.

The two volumes, which may be purchased separately, provide an impressive amount of fresh material on infancy and childhood in different cultures. The material is illuminating and yet provocative, raising many questions which must be answered by us. At times the lectures are like an analytic hour in which the reader will feel they are verbose and tangential, yet it will suddenly fit together like a jig-saw puzzle and provide us with an insight in child development.

The two volumes are "must" reading for all disciplines.

LOUIS L. LUNSKY

LAFITTE, PAUL. *The Person in Psychology: Reality or Abstraction.* Philosophical Library, Inc., New York, 1957. x + 233 pp. $6.00

The author is on the teaching staff of the psychology department of the University of Melbourne. He was graduated in psychology from the University of London. He had held several psychologic posts in the British army before he went to Australia in 1947. By training and experience the author is primarily a counseling or vocational psychologist. Although he denies any professional competence in philosophy (modern theory of science), his book demonstrates that he has read much and thought well about the requirements of a scientific methodology in psychology. He has a good and sound knowledge of psychoanalysis and has had considerable and relevant clinical experience. His book is outstanding and deserves to be studied by anyone interested in improving the scientific status of psychology. He was awarded the degree of doctor of philosophy for this book, which is a logical analysis of the concepts and different methods used in psychology to study personality.

Many individuals and groups are trying hard to create a unified behavioral science. Most of these efforts aim at a super-science which would absorb and synthesize its constituents, psychology, psychiatry, physiology, neuroanatomy, sociology, anthropology, history, education, etc., and which would be described in the terms and formulae of modern logic. If cautious, these efforts usually end in juxtaposing the special sciences without achieving any synthesis. Sometimes, however, those efforts result in reducing the terminology and thinking of all the sciences to be synthesized in the "unified science of behavior" to the simplest and most abstract terms common to all of them. Consequently the specific contribution of each science is markedly reduced if not entirely lost in the process. We can see this in the attempts to unify behavioral science in terms of mechanics or of systems of intercommunication, or neurophysiologic processes. These attempts, which remind one of similar theoretic attempts of the XIXth century, threaten to interfere with the development, if not the existence of psychology. Dr. Lafitte belongs with the minority of theorists who prefer to concentrate their energy and ideas on the development of psychology as an independent science of individuals acting in a social milieu. Psychology should strenghten its own scientific structure and increase its accomplishments rather than wither away as a constituent of doubtful value in a spurious and premature unified science of human behavior. The author discusses what psychology is, what it should not be, and what it better be if it is to be sound and fruitful. There is a strong tendency in all sciences to apply available but not relevant techniques for the solution of problems rather than to make a search for relevant but non-existent techniques. The largest part of this book is taken up with an analysis of the irrelevant concepts or hypotheses, and investigatory techniques, commonly used in modern psychology. Although it is critical, this is not an angry book. The author frequently formulates requirements of a valid scientific approach to personology. In fact, the author's grasp of the logical

requisites of a valid science of the acting human person is better than his knowledge of what is being done in the world. For example, his analysis of the methodologic foundation of valid projective methods of personality is sound despite the fact that (as is apparent in his description of the projective tests, especially of the Rorschach) he has seen no good Rorschach work; he thinks that the Rorschach data are generally interpreted in a subjective, impressionistic "intuitive" way. Few authors dare to write about the subject-matter with which this author has dealt. Still fewer are capable of throwing light on complexities in terms of what is actually observed during psychologic investigations of individuals without making the concepts more abstract and less meaningful. Not all of the author's remarks are of equal depth or degree of novelty and not all will be acceptable to critical readers. However, by far the greatest part of the book is sound and convincing. It deserves wide reading. Should there be another edition, it might be advisable to expand some of the arguments and make them more discursive in order to make them more persuasive. This is a very thoughtful and thought-provoking book in an area of basic significance: what makes psychology scientific and fruitful.

ZYGMUNT A. PIOTROWSKI

JERSILD, ARTHUR T. *The Psychology of Adolescence.* Macmillan Co., New York, 1957. xii + 438 pp. $5.00

This book by a professor of education in Teachers College, Columbia University, has been "written for students, teachers, and parents, for young people who are still in the adolescent period of growth, and for older persons who wish to inquire into the bearing of their own adolescence on their lives as adults." Addressing himself to this wide audience, the author explains simply, clearly and briefly, the fundamentals as well as numerous other aspects of the psychology of adolescence. He covers the ground well and conscientiously, relying both on observational, statistical surveys of overt behavior and on modern psychopathologic theories of personality. The bibilography is extensive, 35 pages, and there is at least one reference to each bibliographic item in the body of the book. A subject index and a separate name index facilitate the use of the book as a reference text. The author maintains a consistent mental hygiene outlook, pointing out neuroticisms but emphasizing also under the influence of the existentialists that growth is painful and conflicts are unavoidable. The book keeps clear of controversial issues and limits itself to what is currently accepted as valid. It is strongly influenced by psychoanalysis but omits Freud's ideas about the influence of infantile sexuality upon the character structure. Like most modern texts on psychopathology as well as on psychology, this book does not discuss the unconscious. There are several references to the unconscious but the latter concept as used by the author, means simply "unawareness." A number of Kinsey's data are reported in a tactful way, not likely to offend anyone. The book reads very well and has been written with a sense of responsibility. It certainly belongs among the leading textbooks on the psychology of adolescents and can be used in colleges and perhaps even in high schools.

ZYGMUNT A. PIOTROWSKI

JACOBI, JOLANDI. *Komplex, Archetypus, Symbol in der Psychologie C. G. Jungs.* Rascher Verlag, Zurich, 1957. xi + 223 pp. ca. $4.00

This is not Dr. Jacobi's first book presenting an unhurried, clear, systematic, and teacher-like exposition of C. G. Jung's ideas. This introduction into Jung's basic concepts of the complex, the archetype, and the symbol is lucid. The three concepts are the most characteristic of Jung's psychology; it is in their use and definition that Jung differs most from Freud. By means of appropriately chosen excerpts from Jung and helpful com-

ments of her own, Dr. Jacobi has demonstrated that these basic concepts have been adequately defined and can be understood provided one takes the trouble to find out what is meant by them. However, as in the case of other psychologic concepts offered by other authorities in the field, clarity and consistency of definition do not always guarantee an easy and simple application of the concepts in individual cases. It is quite apparent from a comparison of Jung with Freud that, in their theories of personality development, Freud emphasized past experiences, going back to early childhood (the Oedipus complex), while Jung stresses not only past experiences, but also psychobiologic forces of growth, active in the present. Although the followers of Freud and Jung tend to see an unbridgeable gulf between themselves, modern developments make it gradually more possible to reconcile if not to synthesize the two versions of analytic psychology. With the increasing interest in the defense mechanisms of the ego and its "healing" or "conflict solving" capacities, and the simultaneous deemphasis on early childhood traumata and on the unconscious, more and more attention is being paid to the patient's own attempts to help himself through creative effort at self-development. In passing, it might be noted that archetypes and the collective unconscious refer to universal psychologic problems, common to all human beings, such as love and hate, birth and death, mating and parting, male and female, etc. Dr. Jacobi has written a non-polemic exposition of Jung's definitions of the complex, archetype, and symbol, and has accomplished her purpose extremely well. This readable and thorough little book, with a brief foreword by Jung, can be recommended to both Jung's followers and critics, in fact to all readers who are intrigued by the three controversial concepts and seek clarification.

ZYGMUNT A. PIOTROWSKI

ROHDE, AMANDA R. *The Sentence Completion Method: Its Diagnostic and Clinical Application to Mental Disorders.* Ronald Press, New York, 1957. xii + 301 pp. $7.50

The Sentence Completion technique has been in use since 1879. At first, its purpose was to study intellectual level and intellectual peculiarities. Binet included it in his intelligence test in 1904. It was applied to the investigation of emotional attitudes for the first time apparently by Payne in 1928. Dr. Rohde, now senior psychologist at the Camarillo, California, State Hospital, has been working on her version of the SCM since 1940. The SCM is a variation of the free association method. Instead of single word stimuli, the SCM employs chiefly groups of words which are presented to the subject as beginnings of sentences with the request that he complete the sentences. Dr. Rohde's SCM consists of 65 items which are followed by this written request: "Write below anything that seems important to you". Sample items are: The future, I feel, If I, Eating, God, I am very, Death, Love in my life, My father, No one. The opening words are intentionally vague to allow for the greatest variety in completing the sentences, and thus for greater ease in self-expression.

On the whole, sentence completion tests have a very limited value as devices for the detection of intellectual or emotional deviations. Nevertheless, they are frequently used, probably because of the simplicity of administration. The patient can be left alone with the test, giving his responses in writing, and the scoring can be quick and easy. This book differs from other SCM texts and tests chiefly because of the extensiveness of the norms and the relatively large number of sample tests with their interpretation. The author used Henry Murray's scheme of needs and presses to classify the responses to her own SCM. The frequency tables are in terms of needs (*e.g.*, achievement, succorance, defensiveness) and not of type of responses to specific items. The groups of mentally disordered patients were found to differ from the group of "well-adjusted" subjects in a number of these

psychologic needs but these group differences are too few and too small for dependable diagnoses in neuropsychiatry. As a matter of fact, the SCM and other tests which give the subject an opportunity to predict with validity the conclusions that can be drawn from his responses give better results when used on intelligent, cooperative, not very anxious, self-confident and rather healthy subjects than they do when used on the mentally disturbed. It takes some strength to tell the psychologic truth about oneself. Most mental patients lack that strength, at any rate at first contact and when asked to leave a written record of their attitudes. There are some patients who are so poorly controlled and so detached that they give striking responses, but in cases of this kind, the diagnosis is not a problem. The author's own wide experience with her test, reported in this book, suggests that this SCM increases in fruitfulness the more cooperative and normal the subject is. This test takes a leading place among other SCM because of its diverse standardization investigations. It can be very helpful as a first, easy, and quick way of weeding out very disturbed individuals. For pathologic responses are very significant and valid although absence of such responses in a SCM record is not necessarily an indication of mental health. Dr. Rohde offers a detailed and carefully written book on a simple instrument which can be very helpful for comparisons of different groups of subjects despite its limitations in the study of individuals.

ZYGMUNT A. PIOTROWSKI

GRUHLE, HANS W. *Verstehende Psychologie (Erlebnislehre): Ein Lehrbuch*, 2nd ed. Thieme Verlag, Stuttgart, 1956. xi + 633 pp. $11.40

This is the second and revised edtition of a graduate textbook. Not only is this book thick, but its format is large. The author describes his method as one of understanding psychologic processes through empathy, and adds that the methodology of psychology must be basically different from that of the natural sciences. He quotes W. Dilthey approvingly who said: "We explain nature, we understand mental life." Gruhle does not have any definite and comprehensive psychologic theory, and does not attempt to include many diverse phenomena under one theory. He tackles almost every problem, small and large, independently of the other problems. This is not a textbook for the modern psychologic professional who wants to solve research problems and is in search of new methods, or who desires to perform a definite service for others. This is rather the non-experimental nineteenth century type of psychology for the educated and cultured layman who likes to deepen his sensitivity to everyday psychologic phenomena and enlarge his intellectual horizon. Gruhle's psychology fosters observation, speculation, and some degree of understanding of the mental functioning of others, especially the conscious processes. The book will be appreciated most by those who, being interested in human nature, prefer to study it in a leisurely manner for the purpose of improving themselves as civilized members of society rather than of acquiring specific professional skills. Gruhle acknowledges that there are other methods in psychology beside his own which is a combination of direct observation, intuition, reflection, and library research. He grants that the other methods have investigated other important aspects of psychology (personality) and have obtained results inaccessible to his "understanding psychology." However he feels that the emphatic understanding psychologic approach is preeminently psychologic.

Gruhle expresses most of his opinions without hesitation or qualification. He argues with those with whom he disagrees (and he disagrees frequently and with a great many writers). Stating his case plainly, he gives the reader an opportunity to evaluate the arguments of both sides. Many a popular misconception and superficial generalization are pointed out and corrected. Most readers

would agree with Gruhle on most points. However he sometimes omits mentioning good arguments which could be cited in support of a thesis Gruhle opposes, and his numerous criticisms are at times facile and not compelling. The author has read very widely but he quotes professional literature less extensively than the opinions of great writers, dramatists, and philosophers; he quotes literary sources of all times, from ancient India to modern Western publications. The diversity of topics is greater than in any other textbook. Some of the topics are: Napoleon's personality, psychology of art, varieties of religious ecstasy and mysticism, delinquency, thought processes, affect, bullfight from the toreador's viewpoint, superstitions, development of language, symbolism of colors, dreams, politics, genius and madness, psychology of nations, war, etc., etc. The author is a psychiatrist by training. His first book (on juvenile delinquency) was published in 1912. In the thirties he wrote rather extensively and well on schizophrenia. The present book adds to one's liberal education. The author takes the reader across centuries and civilizations, and succeeds in demonstrating the continuity and the gradual development of psychological concepts and theories concerning personality. In this seems to lie the greatest worth of Gruhle's book.

ZYGMUNT A. PIOTROWSKI

TOMKINS, SILVAN S. AND MINER, JOHN B. *The Tomkins-Horn Picture Arrangement Test.* Springer Publishing Co., New York, 1957. xvi + 383 pp. $10.00

This type of test has been used since the nineteenth century as an intelligence test, *i.e.*, for the purpose of measuring consciously controlled rational thinking. Tomkins' and Horn's new version of the test was designed to measure personality traits, especially emotional and social attitudes. Their PAT consists of 25 sets of three pictures. Some of the pictures occur in more than one set, and the same "hero" (a man) appears in every picture. The pictures of each set are printed in a circle. The subject's task is to indicate the most reasonable sequence; he is at liberty to arrange the pictures as he pleases to achieve "that arrangement which you feel makes the best sense." The subject is also requested to write a sentence about each picture to explain his arrangement. *E.g.*, the man is working at a machine; he hurt himself; he is now in a hospital. An attempt was made to obtain measures of "social orientation" (avoidance of or seeking out others, aggression, dependence, lability, sense of duty), of optimism-pessimism, and "of the relative strength of thinking, phantasy, affect." These are aims worthy of any ambitious personality test. However, the PAT is a paper-and-pencil group test and like other personality tests of this kind, allows few meaningful conclusions which, moreover, are of low reliability. The authors themselves report that on a re-examination the scores of the individual subjects varied a great deal but that the group averages changed hardly at all. How then can PAT be helpful in evaluating personality traits of either single individuals or groups of individuals? Tomkins and Miner admit that "the test has the characteristics of an intelligence test." This is not surprising since the pictures present scenes from every-day life, since the pictures are structured and unequivocal, and since the instructions call for arranging pictures in "the most reasonable sequence." All this encourages accurate sensory perception and controlled objective thinking, while it discourages free subjective association and a display of creative imagination. The history of personality testing during the past forty years offers two clear lessons. One is that the scope and validity of a personality test improve with an increase in the degree to which the test stimulates free and creative imagination; therefore, the use of inkblots is more productive than that of pictures of real people in real life situations. The other lesson is that the more a test stimulates logical and

consciously controlled thinking about external aspects of perceptible reality, the less it reveals about non-intellectual personality traits; thus, *e.g.*, schizophrenics whose intelligence test performances agree even in details may differ conspicuously from one another in their personalities.

The PAT allows very little play of free imagination and requires far too much detailed sensory perception and conscious thinking to be a good personality test. A survey of the statistical tables in which this book abounds demonstrates the PAT's limited usefulness (1500 normals and 755 patients were tested). On most paper-and-pencil personality tests, the schizophrenics usually manage to obtain a "more normal" score than do the normal subjects. The PAT also has its surprises: "The organic (cerebral) group is distinguished by high sociophobia . . . With the organics they (neurotics) share non-labile affect, a characteristic which is in sharp contrast to the labile affect of the manic-depressive group . . . Perhaps the most puzzling finding is the high work interest in the (hospitalized) psychotic population." The latter statement contrasts with the authors' finding that the average schizophrenic required approximately three times as much time as the average normal subject to complete the PAT, and that many schizophrenics (in one group, 75 per cent) refused to take the test though they had nothing else to do. The manner in which personality traits were deduced from the test data resembles greatly that described by Tomkins in his monograph on the Thematic Apperception Test. In order to find a meaningful application for the PAT, the authors decided to "discard most of the responses, and utilize for diagnostic purposes only the rare responses . . . What we have gained in certainty and reliability by this method (of using only those responses which occur rarely) we have lost in efficiency. We might by such a method test a million people before we found one about whom we were willing to make a diagnosis. This is, we think, a necessary consequence of our method." The book contains no statistics regarding the percentage of cases about which the PAT permits meaningful statements to be made. The inclusion of an illustrative individual case also would have been helpful. The authors state that the alternative to their test procedure, applicable only once in a long while, is "relying upon the sensitivities and special competence of the experienced clinician." One could think of other alternatives, *e.g.*, the use of plainly stated and reliably applicable rules of interpretation of personality test data (Rorschach, TAT), greatly reducing the subjectivity and reliability in personality test interpretations. It is understandable that the authors should try to save as much of their great labor as possible. They do this mainly in the chapter on the test's "rationale." A number of points in this rationale could be challenged. But the rationale was written *ex post* and, whatever its nature, does not affect the value of the test. However, the authors attempted an impossible task: the creation of a valid and useful paper-and-pencil automatically scored personality test. The work on the PAT was begun in 1942. At that time (and particularly under the pressure of war needs) many psychologists still believed that paper-and-pencil personality group tests of high validity could be constructed. This belief may explain why the work was undertaken.

ZYGMUNT A. PIOTROWSKI

MASSERMAN, JULES H. AND MORENO, J. L., Editors. *Progress in Psychotherapy*, Vol. 2, Anxiety and Therapy. Grune and Stratton, Inc., New York, 1957. 248 pp.

It has long been a matter of common knowledge that, since the time when Sigmund Freud astutely recognized the significance of anxiety as a concomitant of certain psychic disturbances, this phenomenon has been made the subject-matter of extensive investigation and speculation, and of well-